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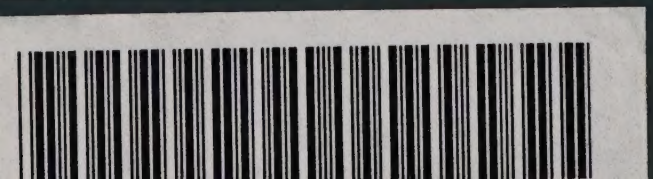

CLINICAL MEDICINE:

Lectures and Essays.

From the Author

Established 1778

CLINICAL MEDICINE:

Lectures and Essays.

BY

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TO

WILLIAM STOKES, M.D.,

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ETC., ETC.,

THIS BOOK IS INSCRIBED

IN GRATEFUL REMEMBRANCE OF THE CLINICAL TEACHING

RECEIVED AT THE MEATH HOSPITAL

BY HIS FORMER CLINICAL ASSISTANT

THE AUTHOR.

MEDICAL SOCIETY OF LONDON
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PREFACE.

THE Lectures and Essays which compose this book have previously appeared in the Medical Journals. The dates and places of original publication are given in the Table of Contents.

A larger experience and a more lengthened study of the subjects treated have, however, enabled the author to make many additions to the original articles. In this way a considerable amount of new matter has been introduced.

The author is glad of an opportunity of thanking those gentlemen who have acted as his clinical clerks during the last fourteen years, and with whose assistance the materials forming this volume have been collected.

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I.

ON THE TREATMENT OF ULCER OF THE STOMACH.

Gentlemen,

We have recently had an opportunity of studying together the treatment of ulcer of the stomach. In to-day's lecture I propose to lay before you, more fully than I have been able to do at the bed-side, my reasons for treating these cases by mechanical and physiological rest. In the first place, however, it may be profitable to review briefly the state of the diseased organ, and the conditions which favour the development and extension of ulceration. The earliest step in the production of gastric ulcer, is the arrest of the circulation in some little vascular territory of the mucous membrane. Such arrest often occurs during catarrhal inflammations, and is followed by extravasation of blood into the tissue, and afterwards by erosion of its surface. Sometimes, degeneration of the small blood vessels is the antecedent condition, but more commonly there is

embolic plugging of some small arterial branch, or thrombosis of a small vein. In either case the process of extension is the same : self-digestion of the spot in which the arrest of circulation has occurred. Dr. Pavy, in an admirable series of experiments,[1] has demonstrated that whenever such an arrest of circulation is produced, the gastric juice corrodes the affected portion of mucous membrane. Under normal conditions the mucous membrane is protected from the solvent action of its own acid secretions, by the constant flow of alkaline blood through its vessels. For a long time it was a puzzle to physiologists, how the stomach resisted the action of the gastric juice. Some referred this immunity to the vitality of the tissue, others to some special property of the epithelial lining, and its constant reproduction ; but modern experimental inquiry in this, as in many other cases, has replaced the vitalistic hypotheses of our predecessors, by a simple explanation, based on well known chemical and physical laws. By a beautiful arrangement of the blood vessels, the blood which supplies the acid constituents of the gastric juice, and which consequently becomes highly alkaline, circulates afterwards at the surface of the stomach round each glandular orifice, and thus opposes, by its alkalinity, the

(1) *Philosophical Transactions*, 1863.

acid secretions in contact with the mucous membrane. By this contrivance the mucous membrane is protected as long as the circulation is perfect; but whenever the blood flow is arrested, the acid secretions, unchecked by the constantly renewed alkaline blood current, begin to digest the surface. In the same series of experiments it has also been shown, that self-digestion of the stomach may be artificially accelerated by rendering the gastric juice abnormally acid, and thus destroying, from the other side, the natural balance between the acid juices and the alkaline blood. In disease the same thing occurs, and hyperacidity renders ulcerative action more rapid, whenever the preliminary breach of the surface has been produced. In fact, when once an ulcer is formed, and the normal vascularity of the organ is altered, every digestive act extends the mischief. Whatever amount of repair has been effected in the intervals of rest, by the tissue elements forming the surface of the ulcer, the stomach, Penelope-like, undoes, by its own secretions, in each succeding act of digestion. Can we wonder then that there is in this affection, great irritability of the stomach, that every attempt to take food is followed by intense pain, most frequently confined to a small spot corresponding to the site of the ulcer; pain that only ceases when the stomach has rejected its contents, or transmitted them into

the duodenum? In bad cases this agonising pain goes on from day to day: daily the ulcer is re-opened and extended, till at last, after some fuller meal (Case 2) or some unusual effort (Case 3), a large vessel is opened, or perforation of the stomach wall occurs. In this way an ulcer once formed, either partially or completely disables the stomach; and as the performance of stomach functions is of vital importance to the patient, so the treatment is necessarily of the highest moment. In the act of digestion the following circumstances at once occur to us, as serious obstacles to the healing of an ulcerated surface, viz.:—

1. The movements and changes in volume of the stomach, following the ingestion of food.
2. The mechanical and chemical irritation of the ulcer produced by the food and the gastric juice.

We cannot wonder that the stomach indicates in the clearest and most unmistakeable manner that its surface is irritated by food, and that it refuses with all its energy to perform functions which, in their very process, aggravate the disease that frets it.

Every attempt to digest food in severe cases of this disease, sweeps away, more or less completely, the results of that curative process which has been going on during the previous state of rest, and

leaves the ulcer, if not farther, still often as far from being healed as ever.

The very functions, then, which it is the duty of the stomach to perform in order to maintain life, are opposed to a great extent to the healing process which we have to promote. Do not understand me to say that no ulcer can heal so long as the stomach is called upon to act, for such an assertion on my part would be opposed to clinical and experimental facts; but rather that the process of digestion interferes much, in cases of gastric ulcer, with the efforts made to repair the lesion. Consider how, in the case of a cutaneous ulcer, similar irritation would act; and how quickly the surgeon would set about removing all such obstacles to healthy action. In the pain caused by the irritating substances and the movements of the part, would be discerned the urgent need of rest; and on rest being obtained by the removal of all causes of disturbance, the healing of the surface-ulcer would quickly advance.

Let us bear in mind this great fact, then, in our treatment of internal ulcers. Rest is as essential to repair as it is to growth; for repair is growth, directed, not to development, but to restoration.

We shall find that in all cases of gastric ulcer, our measures, as in the case of the surface ulcer, must be chiefly directed to procure rest for the

part—rest from all mechanical irritation—rest from all physiological action.

The potent influence of rest in the treatment of disease, has only recently received that scientific consideration to which it is specially entitled. Its efficacy as a therapeutic agent has been recognised mainly in the surgical domain of practice, or in the treatment of the diseased condition of parts endowed with functions which admit of a break in the continuity of their action without disturbance to the system at large. In Professor Hilton's eloquent *Lectures on Rest and Pain*, numerous convincing examples are advanced of the good results obtained by the application of mechanical and physiological rest to the treatment of many surgical maladies, and instances are not wanting to indicate its value in the management of cases more especially medical. The influence of this agent in the hands of the physician is, however, necessarily limited by the very nature of the functions of those viscera whose disorders he is called upon to treat. For in the present state of our knowledge, the cessation of function in many of the internal organs means death, so intimately is the continuity of their action linked with the maintenance of life. The failing heart and the worn-out lungs can know no break in the life-long circle of their toil; for as their continuous action means life, so their repose means death.

Nevertheless, even in diseases connected with the functions of circulation and respiration, the influence of rest is not wholly excluded, but, on the contrary, forms in the present day an element of no small importance in treatment. For although absolute rest, even for a short time, cannot be afforded to these organs, yet by diminishing the amount of their labour, and by removing the obstacles to normal action, we gain for them comparative, or, as it may be called, *partial* rest. In maladies of the nervous system, and in those of some of the excreting glands, no small part of our success would be lost were this weapon taken from our armoury: but it is more especially in diseases of the stomach, an organ for which we can obtain *complete* rest, that we find the greatest triumph reserved for this therapeutic agent. This, to a certain extent, has been long recognised, and partial rest for disordered digestion has formed the basis of treatment in the hands of all masters of our art.

The stomach, when seriously affected, teaches this lesson by its own acts; for in the pain caused by the ingesta, and in their rapid rejection, we have an unmistakeable protest against too officious efforts to restore health by the stimulus of food, when the soothing influence of rest is alone required. That the application of this remedy to the management of stomach diseases

should have been early noticed is not surprising, considering that the indications are evident, and good results rapidly follow its use. But it is surprising that rest should have been generally administered in serious organic diseases in such sparing quantities, such infinitesimal doses: rest at once too partial in its nature and too brief in its duration. For some years past the value of a treatment which affords *complete* rest to the stomach in its serious disorders has been increasing in my estimation, and the practical application of the plan to the treatment of ulcer of the stomach and to many other lesions of the viscus has strongly confirmed my opinion. The nature of the digestive act is undoubtedly the great obstacle to the healing process in gastric ulcer. But nevertheless authors have contented themselves with enjoining only that partial rest for the stomach which a limited diet affords. There is abundant evidence that this mode of treatment is in many cases efficacious, and the case of Béclard may be well cited in its favour. That distinguished anatomist recognised clearly the necessity of rest, as complete as possible for the organ, and in treating himself for this affection, limited his diet to such a degree that it barely sufficed to support life. His almost total abstinence met its reward in the cure of his disease, and at his death the necropsy revealed the cicatrix of an old ulcer. By

far the greater number of cases attending at our dispensaries and hospitals as out-patients, are necessarily treated in this way, and thus week after week, and month after month, the wearing pain and the half starvation have to be endured, till a gradual recovery, too often with shattered health, is obtained, or, as often happens, the sudden gush of blood from an ulcerated vessel, or the rapid collapse of perforation, closes the tedious case.

On the other hand, with the in-patients in our charities, and with all who can obtain good nursing, we have, in the judicious combination of *partial* and *complete* rest, the best means of insuring a speedy recovery. During the first stage of the treatment the patient should be kept strictly to the recumbent posture, all food or drink by the mouth entirely forbidden, and nutritive enemata frequently administered; thus we procure for the stomach a long rest from all mechanical irritation, and from all physiological action. In the second stage, by a carefully regulated diet we should afford that *partial* rest necessary to perfect the cicatrisation, and to accustom the new tissue to the stimulus of the digestive act. The indications we have to fulfil in the treatment of ulcer of the stomach are two:—

1. To promote the healing of the ulcer.
2. To alleviate the symptoms.

By giving the viscus *complete* rest, from all physiological action, from all mechanical irritation, we remove the great impediments to the healing process, which consist in the movements of the stomach consequent on the ingestion of food, and in the mechanical and chemical irritation of the ulcerated surface produced by the food and gastric juice. The second indication is also fulfilled: for the pain and vomiting cease, and the hæmorrhage seldom if ever returns; while the patient, placed in the conditions for causing the least possible tissue-waste, is fairly nourished, and in many cases even strengthened, by the use of suitable nutritive enemata.

The two cases you have recently watched with me in the wards I now read to you as illustrations not only of the treatment of the disease, but also as sketches of its symptoms and progress.

Case 1.—J. H.——, aged twenty, married, was admitted into the Hospital on June 20th, complaining of vomiting and severe stomach pain.

History.—She has never been very strong, and has frequently suffered from attacks of indigestion, She was married at sixteen, has had one child, and has miscarried once. Before marriage she worked as a packer in a warehouse, and afterwards she was at a confectioner's, where the work was very heavy. Since her marriage she has not had much hard work, and has enjoyed improved

health, especially whilst suckling. For the last twelve or thirteen months her health has been indifferent, and about four months back she was seized with severe pain in the epigastrium accompanied by headache and vomiting. For several months her food was frequently rejected, and about one month before her admission she vomited several ounces of blood, and about the same time she passed several black, tar-like stools. The hæmatemesis recurred from time to time with the rejection of food; sometimes merely a streak or two, at other times several ounces of blood being expelled. The catamenia have recurred at the proper intervals during her illness, but she has had a leucorrhœal discharge. Her bowels have lately been confined, and she has occasionally been annoyed by hæmorrhoids.

State on admission.—She wears an anxious expression; face pale; lips anæmic. She complains of severe pain over the stomach, sometimes of a dull and burning character, at other times acute and lancinating. Appetite bad; and she fears to take food as it intensifies the pain. The pain is referred to a spot immediately below the ensiform cartilage, where the tenderness on pressure is very marked, but limited to a spot about the size of half-a-crown. Shooting pains extend now and then from this centre all over the epigastric region, especially after food, when the pain is

most severe, and is only relieved by vomiting. The tongue is moist, and covered with a whitish fur; urine healthy; liver and spleen normal in size. The examination of the chest detected no abnormality in the lungs; but an anæmic bruit was heard at the base of the heart, and along the great vessels. She was ordered to keep strictly to the recumbent posture; to have no food or drink by the mouth, except three ounces of milk mixed with half the quantity of lime-water, to be taken to moisten the mouth during the day; an enema of warm water to clear the lower bowel; and afterwards five nutritive enemata daily, of three ounces each, three to be composed of strong unsalted beef-tea, and two of milk and yolk of egg, with ten minims of tincture of opium in each. Ice to moisten the mouth.

On the following day, the nurse having reported that the milk and lime-water were rejected as soon as taken, they were discontinued. The pain in the stomach a little better, as is also the head-ache. The tongue slightly furred.

June 23rd.—No sickness since all food by the mouth has been stopped. Tongue less furred; pain in the stomach less. The patient is not anxious, and admits that she feels better. The enemata have been retained very fairly, only one having been returned.

24th.—The patient feels better, but very weak;

the pain nearly gone; the tenderness on pressure much diminished. She has a headache, and feels very thirsty. The tongue red and rather dry. The rectum slightly irritable, a portion of the enemata having returned with some fæces three times on the day before. Pulse 84.

26th.—Complains of headache and slight nausea. Pain in the stomach much less. Pulse 60; bowels irritable. Ordered twenty minims of tincture of opium, with three of the enemata during the day.

29th.—Has improved daily since the last note. The irritability of the rectum has been checked by the tincture of opium. She has felt no nausea or pain; pressure over the stomach now elicits scarcely any sign of tenderness; headache much better; tongue red, dry, and furred at the edges; says she feels very weak, and desires to eat. Ordered milk and lime-water, of each half an ounce, every two hours, and only three injections daily.

30th.—Much better; tongue clean. The milk has caused neither pain nor sickness. Ordered milk thickened with a little corn-flour, and to discontinue the enemata after July 1st, and then to take one ounce of fried sole for dinner. Pulse 84, feeble.

July 4th.—Much improved; tongue clean and moist; pulse 80, stronger. She has taken milk with corn-flour and arrowroot, also the fish, with-

out any pain or uneasiness in the stomach. Ordered a small piece of chicken for dinner.

5th.—To take the following twice daily:—Citrate of iron and quinia, four grains; infusion of calumba, one ounce.

10th.—The patient is up, and walking about the ward; feels no pain after eating; enjoys her food, and has a good appetite; tongue clean; pulse 80; no tenderness on pressure over the stomach. At this time she was fit for discharge as far as the gastric symptoms were concerned; but she was kept in the hospital a fortnight longer on account of the anæmia, which gradually disappeared, and with it the murmur at the base of the heart.

She showed herself once a month for several months after her discharge, and on all occasions she was healthy and free from all gastric symptoms.

Case 2.—J. J——, aged twenty-two, dressmaker, was admitted into the Hospital for hæmatemesis.

History.—About one month before her admission she applied as an out-patient, stating that she suffered very much from indigestion and a severe pain in the stomach, increased by taking food; she had occasionally vomited blood with her food. These symptoms began, she said, about eighteen months previously, and increased

in intensity up to the time of her appearance at the hospital. Before this illness she had always had good health. She was ordered some powders containing bismuth and powdered opium, and had full directions given her concerning her diet. She was also told to return immediately to the hospital if the vomiting of blood recurred. The hæmatemesis having again appeared on March 20th after a rather full meal, she was admitted on March 22nd.

March 23rd.—Her face is pale and slightly puffy; tongue flabby and slightly furred. She complains of slight headache, and of pain in the stomach and constant sickness. The pain comes on chiefly after eating; it shoots over the stomach, and is also felt very badly in one spot posteriorly in the left vertebral groove. She is afraid to eat on account of vomiting. The stomach will not retain milk even in very small quantities. On examination the pain is found to be situated just below the ensiform cartilage, and to the left side of the middle line. The area of tenderness on pressure is about the size of a half-crown. Pulse 70, weak and small. Heart-sounds normal. Lungs healthy. Liver and splenic dulness natural. Urinary organs healthy. Temperature normal. Ordered to take no food or drink by the mouth; to have, every four hours, an enema composed alternately of milk with yolk of egg, and strong

unsalted beef-tea, with fifteen minims of tincture of opium added to each; to be kept strictly to the recumbent posture; and to have her mouth frequently moistened with water by the nurse.

25th.—The pain in the stomach is still felt, but not so badly. The sickness has altogether ceased. Tongue cleaner and drier; pulse 80. The treatment has been strictly followed, and the enemata have been retained.

26th.—No sickness or feeling of nausea; the pain much better, but the epigastric region as defined above still tender on pressure. Pain in the back better. She feels low, and very hungry. Tongue dry and slightly furred. Her bowels were moved twice yesterday, but not till two hours after enemata. Pulse 68, weak and compressible. Injections to be continued, with ten grains of pepsine added to each.

28th.—Much easier in the stomach; tenderness less. The pepsine has, however, irritated the bowel, and caused the speedy rejection of the enemata. Slight pain has also been felt along the sigmoid flexure. The pepsine ordered to be omitted, and enemata of milk and egg, with twenty-five minims of tincture of opium in each to be given. Pulse 58, weak.

29th.—She feels much better. No pain in the stomach or back, and scarcely any tenderness on pressure. Tongue furred and dry; pulse 68,

stronger. The enemata have been well retained, and the tenderness in the course of the lower bowel has diminished. Ordered half an ounce of milk every two hours, and two injections daily.

31st.—The milk has been retained, and caused no pain or uneasiness. Tongue moist, and less furred. The milk to be thickened with corn-flour.

April 1st.—The milk has caused no pain or sickness. Tongue clean. Scarcely any pain on pressure over the stomach. The milk and corn-flour to be continued.

2nd.—Tongue slightly furred, but she has had no pain in her stomach, and has enjoyed her food. The enemata, having irritated the bowels slightly, were ordered to be discontinued, and warm poultices to be applied over the lower part of the abdomen. One ounce of uncooked mutton, chopped very fine, to be given in three portions during the day; also two ounces of sherry.

3rd.—Very much improved. The meat has agreed very well. Ordered to continue it in gradually increasing quantities.

5th.—Much improved; tongue clean; no pain or sickness after food; no pain on pressure; appetite good. The patient expresses herself as almost well. Ordered a little of the breast of a chicken, with bread and two ounces of sherry.

10th.—Discharged as quite well. She was directed to be very careful in her diet for a few

weeks, and to return most gradually to her ordinary food. She came to the hospital several times after her discharge, and was quite free from all her former dyspeptic symptoms, and had returned with comfort to her ordinary habits of life.

These cases are fair examples of the milder forms of gastric ulcer which you will commonly be called upon to treat; but sometimes the malady assumes a much graver form, and even the treatment by complete rest for a time is broken by repeated attacks of hæmorrhage from the stomach. In such cases you will require more aid from opium than in the simpler cases, and the period of complete rest will have to be prolonged. In such cases never doubt the efficacy of rest to save your patient, be steadfast in carrying it out thoroughly, and I can promise you that your faith will be rewarded by success. Over and over again it will occur to you to have to recommence the treatment when all seemed going on well, and many times will you have to persist in the treatment when the entreaties of the patient and the doubtful looks of nurses and friends strongly tempt you to give medicines and food by the mouth to check the copious blood flow or restore the failing strength. In no case should you yield. I have never yet seen a case of ulcer of the stomach prove fatal in which this treatment has

been adopted, and I have treated all my hospital and private cases in this manner for some twelve years. On the other hand I have seen patients die of this disease, whom I believe the treatment by complete rest would have saved. The following case, which occurred not very long since in the hospital, illustrates what I have said respecting the severer forms of this malady.

Case 3.—Sarah P——, aged twenty-one, single, for the last year or more has suffered from indigestion, which has lately assumed the form of severe pain in the stomach and in the back between the shoulders, coming on about half an hour after a meal. The pain at times has been very severe, and has been very commonly relieved by vomiting. For three weeks before admission the pain and sickness had increased. On the morning of December 13th she was going by train to her work, and being rather late she ran some distance, but nevertheless missed the train. She went into a friend's house to rest, and had a cup of tea, when the pain came on immediately, and she fell down in a fainting state, and vomited a large quantity of blood. She was brought to the hospital and admitted in a state of great exhaustion. After she was placed in bed she rallied, and complained of great thirst: she was ordered to have no food whatever

by the mouth, but to moisten her lips and tongue with ice and ice water, to have a nutritive enema every four hours, composed alternately of milk ʒiij., or strong unsalted beef tea ʒij., and brandy ʒij.: 10 m. of tr. opii. to be added to each. Patient to remain perfectly quiet in bed.

This treatment was continued for five days, but in spite of the strictest orders she swallowed a quantity of iced water, and had five to six attacks of vomiting each day. The fluid rejected was clear water with a *decidedly acid reaction*. On the sixth day she had some visitors to see her, and in consequence of the excitement she vomited after their departure some 20 oz. of fluid which contained a large proportion of blood. Twice afterwards was this vomiting of blood repeated, for the patient was a very troublesome one, and repeatedly disobeyed orders as to swallowing water. On each occasion between 20 and 30 oz. of fluid, containing much blood, was rejected. At the date of the last and fourth attack of hæmatemesis she had been twenty-one days under treatment, and the bowel had become very irritable, while the dry and raw condition of the tongue gave her great discomfort. The epigastric pain was also very severe. Under these circumstances I ordered injections to be given with a long tube, and to be thrown high up into the bowel. In this way larger injections,

ranging from half a pint to a pint, were retained without discomfort, and with great benefit to the general state. Night and morning eight minims of the solution of bimeconate of morphia were injected *sub cute*. A small blister was applied over the seat of the epigastric pain, and afterwards dressed with morphia. For the dry, raw, and fissured condition of the tongue she found much relief from painting the tongue and mouth with a mixture of bismuth, glycerine, and water. The hypodermic injections of morphia soon made her less restless, and she was kept perfectly quiet under the influence of the drug for eight days. She was supported during this time by the large injections given with the long tube three times, and after the second day, twice, daily. On the ninth day she was allowed to swallow during the day four ounces of milk and lime water (equal parts), and twice, a few minutes before taking some of this, she took ʒss. of the following mixture:—Bismuthi subnit. gr. lxxx., Sodæ bicarb. gr. lxiv., Acidi hydrocyan. dil. m. xxiv., Mucilag. Trag. qs. Aq. ad ʒvi., ʒss. bis in die st.

After the twelfth day an injection was given only once a day, and the morphia *sub cute* only at night. Arrowroot, corn flour, sago, and rice were gradually added to her milk diet, and on the twenty-fourth day a little boiled sole was allowed. While her stomach was getting accustomed to the

stimulus of food several slight attacks of vomiting occurred, but no other bad symptoms interrupted her progress. The stomach pain had entirely ceased, and she steadily gained strength. The anæmia, however, was evident for weeks after her discharge from the hospital, but eventually quite disappeared. When I last saw her, some eighteen months after her illness, she was in blooming health, married, and a mother.

In all severe cases when the intense pain and tenderness point to the possibility of perforation, when the irritability of the stomach causes all food to be rejected, or when hæmorrhage indicates quickly advancing ulceration, I would have you closely follow the treatment I have described. By so doing you will obey the indications of Nature, and cease to irritate an injured organ by forcing it to receive food, to which it as plainly as possible expresses its repugnance.

The most rational and most successful treatment in such cases is to give the *most complete rest possible to the affected viscus by stopping the supply of all nutriment by the mouth, and supporting the patient for several days by nutritive enemata.* Perfect quietude in the recumbent posture must be observed; the lips and tongue should be moistened from time to time with a little water; and everything likely to excite the patient avoided. The

body, thus placed in a condition requiring the least expenditure of material, is easily supported for several days by enemata alone, even when the weakness of the patient makes the treatment seem hazardous. For eight or nine days the patient may be kept, if desirable, on enemata—even longer, if necessary; and during this time the pain, the irritability of the stomach, and of the system, cease; and the sufferer enjoys ease to which he or she has long previously been a stranger. Far from becoming weaker, patients in general rally somewhat while under this system.

When I first determined to try this method of treatment, I expected that great difficulty would occur in obtaining the co-operation of the patients; but I have found in all cases, with scarcely an exception, not only a willingness to submit to it, but scarcely a complaint during its continuance. The pain is generally so intense, and the patient, if hæmorrhage has occurred, so alarmed, that any treatment is willingly adopted, especially one the *rationale* of which is so easily understood.

In private practice I always explain to the patient, or the friends, the reasons which induce me to propose this treatment; and thus, instead of unreasoning opposition, I obtain a hearty and intelligent co-operation. Not long since two very interesting cases occurred to me—one in the practice of Dr. Harvey, of the Lozells, and the other

in the practice of Mr. J. B. Jackson, of this town —in which I felt bound to recommend *complete rest* for the stomach, in consequence of repeated and copious hæmatemesis. In both cases the difficulties as usual vanished when the reasons for using enemata were explained to the patients, who rendered us willing and invaluable assistance in carrying the treatment to a successful issue.

The substances which I at first used for enemata were milk and strong unsalted beef tea and the yolks of eggs, with a little brandy, and a few minims of tr. opii. In one of the cases, following out a suggestion offered by Dr. P. Sonsino, of Florence, in a review of a paper of mine upon this subject,[2] I endeavoured by the use of pepsine to administer animal food partly digested. The first crude attempts failed, but since then I have succeeded with artificially digested meat. The best formula for nutritive enemata has, however, been propounded by Leube. He prepares them by chopping up five to ten ounces of meat very finely, and adding one-third its weight of finely minced pancreas (pig or ox), free from fat. The mixture is to be treated in a mortar with five ounces of luke-warm water, and reduced to a thick soup.[3]

The advantages of these enemata are that they

(2) *Imparziale*, November 16, 1865.

(3) *Practitioner*, August, 1872.

offer aliment already partly digested and capable of absorption; they hardly ever produce diarrhœa or irritation of the intestine; they satisfy the patient, and allay hunger.

In all cases I would have you wash out the lower bowel well before commencing the treatment. Use injections of small quantity, never exceeding four to six ounces, unless, as in Case 3, a long tube is used, and the nutritive matter thrown high up. When irritation of the intestine occurs in spite of opium, rest the bowel for eight to twelve hours. Feeding your patient according to these rules, you may gain for the stomach rest for several days—generally six to ten. After this the symptoms will have so much abated that the treatment by partial rest can be adopted with success.

This interval of *complete rest* from all irritation —from all action—I consider of the greatest importance; and I have found it so beneficial in its results, that I would recommend you to treat all severe cases of this disease, whenever possible, by this plan. The ordinary method of partially resting the stomach by regulating the diet only partly fulfils the indications that present themselves, and in my opinion, and in my experience, renders the time occupied in the repair of the ulcer much longer. In the treatment by *complete rest* for a time, the patients rapidly advance

towards health, and only remain under observation for about three or four weeks. A fortnight of restricted diet, after a week of perfect rest, is usually sufficient to restore the subject to comparative health. In all cases, however, it is essential to insure, as far as lies in your power, a favourable form of diet for some time longer.

By strictly regulating the diet of our patients we procure *partial rest* for the stomach during the completion of the healing process, and thus insure its steady progress. By this method of *partial rest* we can, aided by the drugs I shall speak of hereafter, often cure the milder cases of gastric ulcer, such as you see treated amongst out-patients.

We must regulate, however, both the quality as well as the quantity of the food, and prevent any distension of the organ, while we guard against any irritation of the diseased surface. Simple and well ascertained physiological laws must guide us in our selection of food. We must recollect that certain forms of nutriment are more easily digested than others; and also that some substances—*e. g.*, protein compounds—specially require gastric digestion. Experience teaches that all food which requires much time and energy for its digestion, or which is chiefly transformed in the stomach, proves the most irritating

to our patients. Animal foods, substances likely to irritate the viscus, hot ingesta, and particularly full meals, must be carefully avoided, as they aggravate the patient's sufferings, and are frequently rejected. On the other hand, as we naturally might expect, bland food of an easily digestible form, cool, and in small quantities, is easily borne. In milk we have a fluid meeting all the requirements of the system, and at the same time possessing the qualifications above mentioned—at once the most easily digested and the most elaborately composed food. Sometimes, however, when taken alone, it is rejected; but if diluted with half its bulk of lime-water it is more often retained. This addition prevents more or less the action of the gastric juice upon it; and thus the milk is often passed into the duodenum, to be there digested. By regulating the quantity, however, we can usually insure its favourable reception; and in many cases we find that the addition to the milk of the purer forms of starch, so as to increase its consistence, renders it much more grateful to the stomach. Arrowroot is the best form of starch to use at first. As the patient improves, biscuit-powder, corn-flour, sago, tapioca, and rice may be substituted; and thus the advance to more solid food most jealously guarded. The more easily digested forms of fish may be next allowed; and from these a most gradual

return to the more ordinary forms of nutriment may be made. The greatest nicety of judgment, as well as the firmest control over your patient, must now be exercised; for a single excess will often produce a most serious aggravation of the symptoms. For some weeks after apparent convalescence must you guard your case, and the necessity of this is very evident, when you consider how easily the newly formed tissue covering the ulcer may be irritated.

I may here mention to you a case which, although not one of gastric ulcer in the ordinary acceptation of the term, nevertheless called for much the same line of treatment. I allude to a man who was poisoned by a corrosive fluid (acid solution of chloride of zinc), and whom I treated some years ago in the Queen's Hospital. Gastritis and ulceration arising from causes like this you will often meet with; and they can be best treated by *complete rest*. This man had marked symptoms of gastric injury after he had recovered from the immediate effects of the poison. He was treated for some time on the restricted diet plan. The symptoms varied from time to time; but at last, after rather too full a meal, a severe attack of hæmatemesis occurred. Now thoroughly alarmed, he was glad to assist in any treatment that might prove beneficial, and for six days was fed by nutritive enemata given every

two hours. From day to day he improved; the pain, the tenderness on pressure, disappeared; no hæmorrhage recurred; and when, after his long fast, he began his slight milk diet, no unpleasant symptoms followed. The patient made a speedy and uninterrupted recovery.

Cases of this kind, gentlemen, and the cases of gastric ulcer which you have seen treated in the hospital, speak more strongly and effectually than any words of mine can in favour of a method of treatment which, by its simplicity and rational character, needs little argument to recommend it.

I have avoided as far as possible mentioning the drugs that are used in this disease, because I believe none of them are essential to us in promoting the first great object, the healing of the ulcer. Certain symptoms, however, arise in the course of the malady; and, as in other diseases, the treatment of these symptoms often forms a great portion of our work.

Rest for the organ has the effect of quickly diminishing the pain and allaying all irritability; and, if the rest be complete, these symptoms rapidly disappear. Perforation is best prevented by the treatment I have recommended; but, when it has occurred, the attention ceases to be occupied by the gastric lesion, and is concentrated on the resulting peritonitis. The hæmatemesis is, however, the symptom that we usually have to

combat; and when it is copious, we often find drugs of great assistance. As we have usually time given us to check this hæmorrhage—for it is seldom immediately fatal—we can take a variety of measures to prevent its recurrence. When it occurs in small quantities, the recumbent posture, and rest as perfect as possible for the viscus, usually suffice to stop it. In most cases, ice in small rounded pieces may be swallowed. When the hæmorrhage is copious, I have usually found a mixture containing tincture of opium and gallic acid (15 to 20 minims of the former and 15 grains of the latter) in water, taken every two or three hours, the best remedy. Oil of turpentine—10 to 20 minim doses—has been strongly recommended. Acetate of lead with opium, given in solution, is also most useful in checking the hæmorrhage. As long as rest is observed strictly, the bleeding does not often recur; and this rest, obtained as I have recommended, will usually be sufficient to prevent the return of this bad symptom.

In treating patients, then, by the plan of *complete rest*, we not only place them in the most favourable position for the cicatrisation of the ulcer, but we also, for the most part, fulfil our second great indication—the alleviation of the symptoms. Certain medicines are, however, useful to us in this disease, not so much as curative agents as auxiliaries to our great therapeutic agent—rest.

Opium has deservedly the highest position on the list, and you will find it in this affection most valuable. Not only in allaying pain, and in soothing the general irritability of the patient, but also by its action as a stimulant, and by checking the waste of the tissues, do we find it most beneficial. Mr. Skey's observations as to its efficacy in the treatment of cutaneous ulcers, are equalled almost by the general praise which it receives from all authors who have written on gastric ulcer. I have usually given it in the enemata to my patients; as thus, in addition to its constitutional action, we gain its soothing effect on the rectum, and prevent that irritability of the gut which sometimes interferes much with the treatment by *complete rest*. In some of the milder forms of hæmatemesis, if you cannot altogether stop ingestion of food, you will find good effect from this drug combined with kino, as in powder of kino with opium, or with bismuth.

Of this last drug *Bismuth* I can hardly speak too highly. You will find it and opium your best friends in the treatment of the milder cases of ulcer. Besides the soothing and protecting effect it may have upon the ulcerated surface, it no doubt checks excessive stomach secretion, and so far protects the raw surface from the gastric juice. It also has a very beneficial action on the accompanying catarrh, especially when combined with

alkalies and hydrocyanic acid. It may be given either as subnitrate or carbonate, in ten grain doses, suspended in tragacanth mucilage.

The *alkalies* (bicarbonates of potash and soda, magnesia and lime water) are very useful in neutralising any free acid in the stomach; they should be given between meals, and in some cases, where there is marked acidity after food, with or shortly after a meal. The experiments to which I have referred respecting the action of free acid on the ulcer, make it clear to you how necessary it may be to prescribe alkalies in combination with bismuth.

Counter irritation.—In one of the cases, you saw me blister the epigastric region over the seat of pain; sometimes this plan, when the blistered surface is dressed with morphia, relieves the pain very effectually. In many cases in which *partial rest* only can be enforced, you will find counter-irritation relieve the pain and vomiting, and aid the cure. Let your blisters be small.

Tonics are needed during the period of convalescence, to remedy the cachexia; though this symptom, for the most part, begins to disappear as the healthy condition of the stomach is restored. You will find the milder preparations of iron and quinine the best. I usually prescribe two to five grain doses of citrate of iron and quinine, with half to a fluid drachm of Liq. bis-

muthi in water, or infusion of calumba. This latter is the most suitable vegetable tonic at our command. In the anæmic state, so often seen after copious hæmatemesis, and in young females, tartarated iron and the ammonio-citrate of iron may be prescribed with benefit, especially when associated with bicarbonate or citrate of potash.

Aperients.—With regard to the use of aperients, I must warn you that they are only necessary, as a rule, in the after part of your treatment. Occasionally they are required earlier, but at all times should be of the mildest kind. Castor oil is the best, and may be given in enema, in which form all purgatives are best exhibited. If pills are required, use aloes, or colocynth and hyoscyamus.

I do not dwell longer on the use of drugs, because my chief object is to impress upon you the value of the treatment by *complete rest*, which is, I am certain, at once the most rational, the most safe, and the most sure which you can adopt in severe cases of ulcer of the stomach.

The researches of Mr. Savory[4] in this country, and the more recent experiments of M. Demarquay[5] in France, teach us how great is the absorptive power of the rectum. In all cases in which the stomach refuses to act, or in which rest

(4) *Lancet*, 1863.

(5) *L'Union Médicale*, January 3, 1867.

is likely to benefit the viscus, it is, I submit, our duty to utilise this power. We find this mode of treatment a powerful aid in combating many morbid conditions other than ulcer of the stomach; and, in illustration, I may specially mention the uncontrollable vomiting of pregnancy. My friend Mr. Baines, of this town, has recently sent me the notes of two cases of this kind, in which he adopted the treatment by *complete rest* with the best results, after all the usual remedies had failed. In one case the patient was fed in this manner for more than four weeks. In many cases, now unfortunately matters of history, the same judicious management would, I believe, have saved life.

It has been often remarked to me, that the treatment of ulcer of the stomach and of other conditions by this practice, can never become general on account of its indelicacy. For my own part, I have always found patients suffering from ulcer of the stomach, both in private and hospital practice, too happy to find any means of escaping from the agonising pain to think for a moment of such an objection, and, when hæmorrhage in any quantity has once occurred, too eager to grasp at any hope of cure. False delicacy soon kicks the beam when the love of life is placed in the opposite scale.

II.

ON CYANOSIS FROM PATENT FORAMEN OVALE.

Early in October, 1862, when seeing my out-patients at the Queen's Hospital, my attention was attracted by two children—a boy and a girl, aged respectively three years and eight months, and two years. These children at once forced themselves into observation by the blue duskiness of their faces, the purple colour of their lips, and their heavy, unintelligent expression. On closer examination, they were found to be affected in different degrees. The boy, darker in colour, and more stupid in appearance, seemed to take no interest in surrounding objects; he was unable to walk, and had never made efforts at speech. The girl, on the other hand, paler in colour generally, but with equal blueness of the lips, evinced occasional interest in passing objects; and when irritated showed more decided signs of anger; she could walk a little, and sometimes made attempts to speak.

The surface in each was of a dull blue colour, much darker on the fingers and toes, which were, however, *well-formed*. The lips were of a dark purple hue, and the mucous membrane of the mouth was equally discoloured. The pupils were dilated and sluggish, the conjunctivæ bluish, with the vessels of the sclerotic large, numerous, and purple. The skin was smooth and soft, but dry, and very cold, especially on the extremities. The tongues were clean, the appetite said to be good, thirst sometimes marked, the bowels usually slightly relaxed, but very much so at present. The stools generally very dark in colour. Micturition not frequent. Urine and fæces very often passed in bed.

The thorax of the boy (Cornelius H.), on inspection, presented a remarkably rounded appearance at the upper part, towards the left side, causing his chest to look somewhat barrel-shaped. His breathing was laboured, and attended with very marked action of the diaphragm. Respirations 38 per minute. Resonance on percussion all over front of chest; slight dulness behind at the bases of the lungs. On auscultation, sibilant and mucous rales were heard pretty generally all over the front and back of chest, but more marked posteriorly. The heart's action, though quick, seemed regular; apex beating between fifth and sixth ribs. The area of cardiac dulness was

slightly increased towards the right side. On auscultation, a murmur was heard with the first sound, varying in intensity at different times, and in different positions of the patient. Heard loudest between third and fourth ribs of left side, at their junction with the sternum; most distinctly with the patient placed in the sitting posture, or leaning forwards. The sound was heard as high as the upper margin of the third rib, but was not heard at the apex. Second sound healthy. Pulse 96, weak and small.

The abdomen was found to be tympanitic. The liver was enlarged, could be felt half-an-inch to one inch below the ribs, and stretched across the epigastric region. No perceptible increase of splenic dulness.

In the little girl (Mary Ann H.) the chest was very fairly formed, with only a very slight increase of roundness in the upper part. The breathing, though much more laboured than natural, was easier than in the other case. Percussion sounds resonant all over the lungs, with the exception of the most posterior and inferior portions. Sibilant and mucous rales were heard over front and back of chest. There was no perceptible increase in cardiac dulness. On listening to the sounds of the heart, a faint murmur was heard with the latter part of the first sound, on a level with the lower edge of the third rib, at its

junction with the sternum. This murmur varied in intensity, and occasionally gave rather the idea of a muffling of the first sound. It did not extend upwards, and was not to be heard at the apex. When the patient was placed on her left side, or leaning forwards, it was more distinct. The second sound was normal. Action of the heart regular; pulse 110, small and weak. The abdomen was slightly tympanitic. The area of hepatic dulness was increased, extending about one inch lower than natural, and considerably towards the left side. No enlargement of the spleen could be detected.

The mother informed me that the children were both much discoloured at birth; the cyanosis was then, however, more marked in the little girl. They are very liable to paroxysms of passion, during which they become much darker in colour, and their breathing more laboured. They have had occasionally attacks of palpitation and difficult breathing, unconnected with passion. During sleep they often have convulsive twitchings of the limbs. They generally have a cough, and expectorate watery mucus. They are always cold; and, although they have had no particular illness, they have always been low, weakly, and as bad as they now are.

The mother's history I found to be as follows:— She was always healthy until a few months before

marriage, when she had a bad attack of rheumatic fever, which from her account, seems to have affected her heart; there is however, at present no trace of disease in the heart, which I have examined carefully. She married, when twenty years old, her first cousin, then aged nineteen. He has been always healthy and steady, has no signs of disease of the heart, and has no trace of syphilis in his history. During her first pregnancy she suffered much from palpitation and pain in her left side. She carried the child only eight months; on birth it presented marks of cyanosis, and lived only eight months. Her second pregnancy was attended by no cardiac symptoms, and the child, born healthy, is still alive and well. The third child was born with marked blueness of the lips and surface, after a gestation attended with pain in the side and palpitation. It lived twenty-one months. The fourth child was born healthy, and is still alive. No cardiac symptoms attended this pregnancy.

The fifth, sixth, and seventh children, at birth, all presented symptoms of marked cyanosis; and these pregnancies were all attended by the heart symptoms. The fifth child lived for six years, but never walked or talked. The blueness of his surface continued, and became more marked towards his end. The cases of the sixth and seventh children have just been related.

The children having been brought to the hospital on account of the looseness of their bowels, were first treated for this by chalk mixture. This soon remedied it; and the bronchitic symptoms were relieved by a little expectorant mixture. I ordered them also such warm clothing as the mother could procure for them, with nourishing diet. On their third visit I prescribed for them cod-liver oil, with the view (as laid down in books)[1] of increasing animal heat. Accordingly I made observations of the temperature of different parts of their bodies, and found that, in the case of Cornelius H., the fingers were often not more than 5° to 8°, the toes 1° to 3° higher than the temperature of the room in which he was placed. In the little girl, the fingers varied from 6° to 9°, the toes from 2° to 4° higher than the surrounding atmosphere. In the mouth (under the tongue) the temperature averaged—in the boy, 96°, and in the girl, 97½°. In the boy it once fell so low as 90°, while the lowest observation in the girl was 92°.[2]

The treatment with cod-liver oil I found did not

(1) *Vide* Walshe on *Diseases of Heart and Lungs.*

(2) These observations were recorded before the Clinical Thermometer was a recognised instrument of research in the hands of the physician, and were consequently made by the aid of an instrument graduated according to the Fahrenheit scale, but much less delicate than those now in use. The fingers and toes were wrapped in cotton wool, and the bulb of the thermometer placed between the fingers or toes, and retained there for fifteen minutes. The figures are only valuable as showing roughly the low temperature at the periphery.

benefit the children by producing any marked increase of the temperature of the surface, which on one or two occasions rose 2° in the boy, and 3° in the girl; but at other observations fell to its usual standard. I therefore determined to try another plan of treatment, and I ordered the children four-grain and three-grain doses respectively of chlorate of potash. This drug I prescribed with the view of oxidizing the blood, by means of the oxygen set free by the decomposition of the salt. When I next saw them their colour was much improved, and their temperature had risen from 5° to 6°. I continued this treatment, giving drachm doses of cod-liver oil in addition, for some two or three weeks, with marked benefit. At this period, however, I unfortunately lost sight of the children for some two months; and on their re-appearance I learned that they had been for some time in the workhouse, and had only just been discharged. The privations that the children had undergone before they had entered the workhouse had materially weakened them; and even now the extreme poverty of the parents rendered the supply of the bare necessaries of life often precarious. In March I determined to try the effects of the new therapeutic agent, peroxide of hydrogen; but as I gave it at first in small doses only —viz., two minims to the younger and four to

the elder, I found it no improvement on the chlorate of potash. I have, however, since given it to the boy in eight-minim doses, three times a day, with marked benefit. Under this treatment (although the disease has evidently progressed in some respects since he was first presented at the hospital, owing to the unfavourable hygienic conditions in which he has been placed) a marked improvement in his colour has occurred: his breathing has become less laboured, and the temperature has risen very considerably, having reached in the upper extremities, almost its natural standard; in the toes the increase is not so great, but is nevertheless considerable.

While I was out of town for some short time in April, the little girl died. It appears that for some days before her death the child laboured under a sharp attack of congestive bronchitis, and her colour became much worse. Slight jaundice came on some forty-eight hours before her death. This, in addition to the cyanosis, caused great discoloration of the surface, making it, as the mother expressed it, "all colours in turn."

On the 22nd of April, the day after her death, Mr. Thompson, one of the resident medical officers of the Queen's Hospital, made the *post-mortem* examination.

The surface of the body was livid, but not so

dark as it had been during life. Body rather emaciated; no œdema of the lower extremities. The lungs were found to be much congested, and frothy fluid exuded freely when they were cut into. No tubercle was found; the mucous membrane of the bronchi was red and congested. The pleuræ contained some few ounces of serous fluid on each side. The pericardium, when opened, was found to contain one ounce and a-half of serous fluid. The heart was normal in appearance and size. Large fibrinous clots were found in the right cavities adherent to the walls. No hypertrophy of the ventricles. Right auricle, if anything, slightly thickened. Left auricle normal. No trace of any disease of the tricuspid, mitral, or semilunar valves was detected by the most careful scrutiny. *The orifices of the aorta and pulmonary artery were not in the slightest degree narrowed;* they were repeatedly examined by myself and others, but no narrowing of either orifice was present. The pulmonary orifice was one-sixth more in circumference than the aortic. The only abnormalities, in short, which could be detected were patency of the foramen ovale, and a rather large-sized Eustachian valve. The communication between the auricles was small, admitting only a large-sized goose quill; it was due to the want of development in the valve of the foramen ovale. This valve, attached above and below in

the left auricle, was deficient in front, leaving an opening of the size mentioned, when the septum of the auricles was in its natural position. Pressure on the valve from the left auricle decreased the aperture of communication, while similar pressure on the right side increased it. The Eustachian valve presented nothing worthy of remark beyond its large size. The ductus arteriosus was closed. Abdomen.—Some little serous effusion was found in the cavity. The stomach was pushed towards the left side by the liver, which was considerably enlarged, and of a pale yellowish colour—in spots almost yellowish green. It did not appear fatty; no microscopical examination was made. The spleen, normal in structure, was not enlarged. The other viscera were healthy. The head was not examined.

An attentive perusal of the history of these two children, and a careful consideration of the *post-mortem* appearances discovered in the little girl will, I think, suggest to the mind several points of much interest. The most notable of these I propose to consider briefly under the following heads:—

I. *The symptoms presented by the integumentary system (general symptoms).*

(*a*). The discoloration of the surface.

(*b*). The temperature of the general surface.

II. *The physical signs found on examining the heart's sounds during life, and the post-mortem appearances observed in the case of the younger.*

III. *The conditions presented in each by the nervous system.*

IV. *The treatment.*

V. *The antecedent history of the children and of the parents.*

I.

(*a*). Discoloration of the surface of the body, such as existed in these cases, and even to a more intense degree, has been observed in many cases without the existence of any lesion admitting of admixture of the arterial and venous blood. Patency of the foramen ovale, and other free communications between the right and left cavities of the heart, have also been recorded in which no blueness of the skin was present. Marked cyanosis has, on the other hand, been frequently observed in cases where the only morbid condition found after death has been either constriction of the orifice of the pulmonary artery, or some other lesion causing great congestion of the venous system.

Armed with these facts, many authors, headed by Morgagni, Louis, Ferrus, and more lately by Dr. Stillé, argue that cyanosis depends, not on the mixture of the venous with the arterial blood,

but solely on venous stasis, produced by contraction of the orifice of the pulmonary artery, or other lesion producing similar effects. Other authors,[2] and with these I am disposed to agree, attribute the discoloration partly to the admixture of the venous and arterial blood, and partly to the congestion of the right cavities of the heart and the systemic venous system, which is the consequence of the lesion permitting the mixture. They at the same time admit that contraction of the orifice of the pulmonary artery, and like causes are, when very marked, capable of producing general cyanosis.

The intensity of the discoloration is also much greater, as pointed out by Dr. Chevers, in cases in which the venous congestion has existed since birth, or from a tender age, on account of the probable greater dilatability and capacity of the vessels in early life. It is highly probable, indeed, that the defective development of the heart seen in many of these cases is associated with a similarly imperfect condition of the blood vessels. The arteries in the case I have recorded were very thin in their coats, and it is quite possible that the fœtal condition of the vascular system continued after birth. Newly-formed vessels are always delicate, thin, and transparent, and it may

(2) Gintrac, Hope, Walshe: *Diseases of Heart and Lungs*, 2nd edition, 1854; Speer: *Medical Times and Gazette*, 1855, p. 412.

be that the persistence of these conditions accounts partly for the dusky colour of the surface, by rendering the distension of the vessels more easy, and the dark colour of the blood more visible. The condition of the skin has also a considerable share in modifying the intensity of the colour. In all cases, I believe, where marked cyanosis exists, the skin is generally thin and dry, and the cutaneous exhalation almost, if not entirely, lost. Now, when we consider that a large amount of carbonic acid, and carbon in other forms, is constantly being given off by the skin in health, and that the stoppage of this respiratory function of the surface has been found in animals to produce asphyxia, we can well understand the important part played in the aggravation of the cyanosis by the arrest of this exhalation.

The thinness of the skin also allows the dark blood filling its capillaries to be more apparent.

In applying these facts to the cases before us, we find, I think, that the view which attributes the cyanosis to the admixture of the blood, as well as to the venous stasis, is more consonant than any other view with the phenomena observed. In the little girl there was certainly no condition found in the heart, except the patency of the foramen ovale, to produce the lividity of the surface. This lesion, I consider, by permitting the passage of the blood from the right

auricle into the left, not only caused the mixture of the blood, but also produced more or less obstruction to the return of the blood from the lungs into the left auricle, and thus caused, secondarily, congestion of the lungs, of the right cavities of the heart, and of the general systemic venous system. The part played by the congestion of the lungs in both these cases, was well illustrated by the fact, that any increase of this condition was always accompanied by greater discoloration of the surface. It has been observed, also, that as long as a full proportion of blood be circulated through the lungs, although this red blood be afterwards mixed with an equal quantity of black blood in the heart, yet no great discoloration of the skin occurs: but the moment any congestion of the lungs arises to disturb these proportions, we have, more or less, marked cyanosis. The condition of the heart in the boy I am inclined to consider similar to that found in the girl, with, however, a larger aperture of communication between the auricles.

The state of the skin in both cases has been spoken of as thin and dry—so dry that the mother says the children have seldom, if ever, perspired. This dryness of the surface, although probably to some extent dependent on the circulation of venous blood in the cutaneous vessels, nevertheless I think, by stopping the exhalation of the

hydro-carbons and carbonic acid through the skin, reacted on its cause, and materially increased the evil. The thinness and transparency of the skin, too, allowed the large and numerous capillaries to be more easily seen.

(*b*). The temperature of the surface of the body is always found to be considerably lessened in cases where the blood circulates in a venous condition. This we can readily explain by the fact that between the imperfectly oxidized blood and the surrounding textures, those chemical changes cannot take place which require for their production blood containing a large proportion of oxygen, and which result in the formation of animal heat. Naturally, then, in cyanosis, where this condition especially obtains, we expect to find the temperature of the surface of the body considerably lower than in health. Hence we find, in the cases before us, a marked want of warmth of the surface. The temperature of the fingers in the boy was often found as low as 64°, and the toes 59°, in a room where the thermometer stood at 58. The palms of the hands and lower part of the fore-arm never showed a higher temperature in the boy than 70°; and on one occasion the temperature in his mouth fell to 90°. The observations in the girl accorded well with those in the boy; her surface, however, as might be expected from the less

marked cyanosis, being generally a degree or two higher. We have in cholera, under somewhat similar circumstances (viz., the circulation of imperfectly arterialized blood, and the almost complete arrest of the chemical changes accompanying the disintegration of the tissues), the temperature as low as 70°, and sometimes even lower. I cannot, however, recall at present any cases of cyanosis in which the temperature was so low as in the case of Cornelius H. This remarkable deficiency of the power of forming animal heat in these cases, is to be accounted for by the conditions under which they laboured, viz.:—

1st. The circulation of insufficiently oxidized blood through all the systemic vessels.

2nd. The arrest of the respiratory functions of the skin.

3rd. The mal-nutrition of the children caused by the deficiency of the supply of nutritive food.

The two former of these conditions were the more marked in Cornelius, in whom the temperature was lower.

II.

In remarking upon the physical signs found on examining the sounds of the heart in these cases during life, our attention is arrested by the cha-

racter, the position, and the variability in the murmur as heard in each. In the boy, the bruit heard synchronous with the first sound was fairly distinct. It was audible chiefly with the latter half of the sound, and was heard loudest over the base of the heart, but was not heard either along the course of the aorta or the pulmonary artery. During the whole period of treatment it has always borne a direct proportion to the congestion of the lungs, and to the discoloration of the surface; and since, under the treatment, the circulation through the lungs has been more free, the murmur has become fainter. In the girl the bruit was never heard with the same distinctness as in the boy, but gave rather the idea of a muffling and slight prolongation of the first sound; but was always louder in proportion to the pulmonary congestion; and when this condition was least the murmur was inaudible. Its position corresponded with the base of the heart. In my clinical observations to my class, after I had observed the cases for some time, I attributed the production of these murmurs to patency of the foramen ovale. This opinion I also put forward in a paper read before the Birmingham Branch of the British Medical Association. I was led to this diagnosis by the want of any evidence of the hypertrophy of the right ventricle, by the fact of the murmur not being traceable along the great vessels, and by the

faintness and occasional absence of the bruit, all of which conditions I conceived to be more or less incompatible with any constriction of the orifice of the pulmonary artery, the usual source of murmurs in these cases. Deficiency in the septum of the ventricles would, I considered, have caused the blood to flow from the left to the right cavity, on account of the greater power of the left ventricle; and any other considerable malformation of the heart would, I presumed, have caused much greater irregularity in its action. My views, as thus expressed, were singularly confirmed on examining the heart of the little girl after death. Repeatedly and carefully I investigated the condition of all the valves and apertures, with the uniform result of finding no other abnormal condition to account for the murmur but the patency of the foramen ovale. The perusal of Dr. Markham's case (*vide Med. Times and Gazette*, April 4, 1857), which presented, in the condition of the foramen ovale, some resemblance to the state found in the heart of the girl, confirms me in the opinion that the modification of the first sound in her case was caused by a murmur generated at the patent foramen. Let us consider briefly the conditions in this case favouring the production of such a murmur. We have a valve stretching across the foramen, and attached above and below in the left auricle, leaving an

aperture, elliptical in shape, between its margin and the circumference of the opening. This aperture would be increased by any pressure from the right side, and proportionally decreased by a similar condition in the left auricle; the current of any flow of blood, therefore, would be rather from the right to the left cavity. The unusually developed condition of the Eustachian valve in this case, would, I conceive, direct the current of the blood from the inferior vena cava towards the foramen ovale, and this flow, impinging upon the body of the valve, would increase the aperture, and cause the valve to assume that tense condition by which would be produced an open passage for the blood from the right to the left auricle. Any congestion of the right cavities would augment this condition by dilating the opening still more, and would thus favour the increase in intensity of the murmur by the flow of a stronger current of blood into the left auricle, while the opposite condition of the right cavities would diminish it. The occurrence of the murmur, too, during the diastole of the auricles, and, therefore, synchronously with the systole of the ventricles, supports this view, as pointed out by Dr. Markham. From these considerations, and also from the fact that repeated and careful examinations of the heart disclosed no other morbid condition capable of producing the bruit, I am constrained to agree with Dr. Mark-

ham in supposing that patency of the foramen ovale can, under certain conditions, produce a murmur with the first sound. The special condition present in this case, on which I am inclined to lay stress, was the existence of a well-developed Eustachian valve, by which the current was directed towards the patent foramen. Dr. Markham dwelt in his paper on the existence of a valve connected with the inter-auricular opening, which, by its tension and vibrations, generated the sound as the stream passed over its margin. The chief factor, however, in the production of the murmur was no doubt the passage of a rapid current of blood under considerable pressure from the over-full right auricle, through the patent oval hole, into the less distended left auricle. The blood stream in passing through assumed the jet-like form called by Savart the *veine fluide*, and produced the murmur by its sonorous vibrations.

The condition of the liver, as found on *post-mortem* examination, is, I consider, worthy of remark, as showing the efforts made by this viscus to supplement the action of the lungs. In all the lower animals a pretty constant relation exists between the activity of the lung functions and the size of the liver. Whenever the lungs are inefficient, and fail to decarbonize the blood, we find a proportionate increase in the size and activity of the liver. In this case, from the

defect of the heart's formation, and in consequence of this, from the inability of the lungs to perform their duty thoroughly, we have, through the circulation of venous blood in the systemic vessels, an increased amount of decarbonization to be effected by the liver, and therefore the hypertrophy. The yellowish-green colour of the organ was due, I think, to its activity, and reminded me of the peculiar colour, described by Weber, as seen in the livers of frogs during the season of their greatest activity. The dark colour of the stools, and the occasional diarrhœa to which the children were subject, were due, I believe, to the large secretion of bile; and the jaundice observed in the little girl, before death, was owing to the arrest of this secretion, from the failure of the liver to respond to the extra call made upon it during the bronchitic attack.

III.

The symptoms presented by the nervous system in cases of cyanosis are not often so well marked as in these cases, according to many observers; some authors,[3] indeed, go so far as to assert that there is usually no impairment of the intellect. In the cases before us, the dull and stupid expression of the countenances of the children was remark-

(3) *Vide* Fuller on *Diseases of the Chest*, article "Cyanosis."

able, and the diminished intelligence was found to be much greater in the case of the elder, in whom the cyanosis was the more intense; while in the girl, whose colour was never so bad, the intellectual development was much higher. All the children thus born (*i.e.*, cyanotic) were, according to the mother, very stupid; and the one who lived to be six years old never spoke or walked. From what we know of the effects produced by the circulation of venous blood through the brain, the want of intelligence, as exhibited by these children, does not appear unaccountable. Sir Benjamin Brodie, in his *Psychological Inquiries*,[4] says, when speaking of the circulation of venous blood in the brain :—"The dark-coloured blood, if it has once been transmitted to the brain, even for two or three minutes, leaves an impression on it, from which it may not recover for half an hour, or even longer." And again, in the next sentence but one, he says :—"In fact, the dark-coloured blood transmitted to the brain operates as a narcotic poison." This recalls to our mind the fact that Dr. Snow laid down the great law that narcotism is but suspended oxidation. It is, from these considerations, natural to suppose that the constant circulation through the brain, of blood deficient in oxygen, must materially affect the intellectual

(4) Page 122, second edition. 1855.

development. In a case, however, published by Dr. Richardson (*vide Medical Times and Gazette*, December 22, 1860), the intellect was remarkably clear. From a perusal of this case, I am rather led to think that the mental clearness was due to the fact that there had been no great interference, during the greater part of life, with the functions of the lungs, and that, therefore, the exhalation of carbonic acid and the absorption of oxygen took place freely. When these conditions are not fulfilled I believe that more or less disturbance of the intelligence will always result, and that in all cases of marked cyanosis the intellectual powers will always bear a strict proportion to the quantity of oxidized blood which reaches the brain. The irritability of temper we may, I think, attribute, with justice, to the disturbance of the brain functions, consequent on the circulation of venous blood; and to the same cause, acting on the cerebro-spinal system, is to be placed, partly, the inability exhibited by the little boy to walk, and not solely to malnutrition of the muscles. The other symptom—viz., the twitchings of the muscles—which so often disturbed the sleep of these children, we may fairly attribute to the circulation of black blood through the muscles; muscular spasms being known to frequently occur under such conditions.

IV.

In all systematic works which devote a chapter to this peculiar disease, the merely palliative character which the treatment is obliged to assume, on account of the incurable nature of the malformation, is justly noticed. But after the recommendation of such remedies as are likely to benefit the paroxysms of difficult breathing, palpitation, &c., we are told "that the temperature of the skin" must be "maintained by warm clothing, moderate exercise, and friction; and that of the body, generally, raised, if the stomach be not disordered thereby, by the free consumption of oil, fat, gum, and other aliments of respiration." (5) In the early treatment of these cases I followed the principles generally laid down, but failed to obtain any satisfactory result with regard to the increase of the temperature of the surface. This is not, I think, to be wondered at. For I cannot see how any benefit is to be derived from the addition of carbonized materials to blood already laden with carbonic acid. The congested state of the lungs, consequent on the malformation, prevents these organs (already overworked) from making any effort to obtain for the blood that amount of oxygen essential for the production of heat by the combustion of such

(5) *Vide* Walshe, *Diseases of the Heart*, page 435, fourth edition.

carbonized food. Such treatment can afford no benefit; for unless we can at the same time guarantee an equivalent increase of oxygen, the additional carbon only aggravates the evil. We cannot look to the lungs to absorb for us their extra quantity of oxygen from the air, and, therefore, I submit, we must either feed the body with this element, by affording to the lungs air containing a larger amount of oxygen than exists in the atmosphere, or supply it through the stomach, by the administration of such drugs as will afford, by their decomposition, free oxygen to the blood. To use a homely illustration—the smouldering embers in the fireplace of an ill-ventilated room are soon extinguished by the free addition of fuel; but by the addition of fuel in small quantities, and the admission of a copious draught of air, the dying ashes are soon kindled into a genial blaze. I adopted the latter of the two alternatives pointed out, in my treatment of these children; and at first tried to fulfil the indication by the administration of chlorate of potash, hoping that by the decomposition of the salt its large proportion of oxygen would be absorbed by the blood. The success which attended the use of this drug was satisfactory; and I continued it for some time, during which period the colour improved, and the temperature rose several degrees. This success confirmed me

in the opinion that this was the rational treatment, and induced me to substitute for the chlorate of potash a more powerful oxidizing agent, the peroxide of hydrogen (HO_2), introduced by Dr. B. W. Richardson. At first, as I gave this remedy in small doses, it acted little better than the chlorate; but from the moment that I gave it to the boy in fair doses, his appearance changed. The colour of the face, which was before very cyanotic, became so much brighter as to be but slightly darker than natural; and his cheeks assumed, in place of a violet blue colour, a dusky reddish hue. His eyes, which before were dull, heavy, and expressionless, cleared and brightened, and followed with evident interest the movements of a stranger, and at once fixed on attractive objects. His breathing before laboured, became much easier; his appetite improved; and the twitchings of the muscles ceased. The skin, before always dry, became soft, and on the body frequently moist. Above all, the temperature rose to be, on an average, between the fingers, 82°; between the toes, 73°; and, in the groin, 91°: on one occasion the fingers were 89°. All the observations were taken in rooms varying in temperature from 63° to 64°. When the mother, from domestic causes, was prevented bringing her child to the hospital, and the administration of the remedies ceased, the child relapsed almost to its former condition. The drug is, however, now

constantly given in eight minim doses, three times a day, with a drachm of cod-liver oil twice a day, with the best effects. The child has improved both physically and mentally. When I last saw him (June 6th) he was playing with some toys, in which he evidently took a lively interest. The cyanosis was scarcely perceptible, the only peculiarity striking the observer being a slight blueness of the lips. His mother told me that he had made attempts, of late, to speak; and he can now utter, with tolerable distinctness, two or three words. His stools are much better in colour. His appetite is good. The liver still continues enlarged and active. The paroxysms of passion still often recur; but his colour is, by no means, as dark as formerly during their continuance. The temperature of the surface still continues to average the same as stated above—the only parts ever feeling cold being the toes. The continuance of the administration of the remedy will, I think, be attended with still further improvement in the child's state, as the blood becomes more normal in its character and circulating through the different organs in a more highly oxidized state, enables them to perform their functions more naturally, and, therefore, more perfectly. The great success which has attended this treatment, and the retrogression in the child's state which marked its temporary cessation, are, I think, the best proofs I can offer of

its physiological correctness. The peroxide of hydrogen will, I doubt not, prove a valuable addition to our Materia Medica. Its action in this case leads me to rank it as one of the best, if not the best, oxidizing agent, we possess for internal administration; and I believe its use will be found to be attended with great benefit in all diseases marked by deficient oxidation.

V.

The antecedent history of the children and of the parents affords many interesting and singular peculiarities. The more striking of these I will merely enumerate. They are:—

(*a*) The near relationship existing between the parents—first cousins.

(*b*) The rheumatic fever from which the mother suffered shortly before her marriage, and which probably affected her heart.

(*c*) The absence of any evidence of heart disease in either parent.

(*d*) The peculiar cardiac symptoms which attended the gestation of each cyanotic child.

(*e*) The number of children born with cyanosis—five out of seven.

(*f*) The production of two healthy children between the diseased ones—viz., the second and fourth—both still alive and free from all signs of malformation or disease of the heart.

POSTSCRIPT.

While the two children whose cases suggested the above remarks were under observation, the mother again became pregnant. During the period of gestation she frequently complained to me of heart symptoms, and predicted that the child she carried would, as in previous instances when she had been similarly troubled, be born blue. I frequently examined her heart, but detected no abnormal sound; occasionally its action was rapid and weak, and once or twice her pulse was slightly irregular. These conditions, however, were not at all constant, and only came on now and then. At the full time she was delivered, and a few days afterwards I saw the child. It was distinctly cyanotic: not so much so, however, as the little boy. I watched the child for some months, and towards the end of the first month the cyanosis became less marked, and when it was two months old there was no trace of it left. Soon after this the parents left Birmingham, and I lost sight of the family. This last case I regard as an instance of recovery from cyanosis, possibly by closure of the patent foramen ovale. In this child's heart no murmur was heard: there was occasional dyspnœa during the period the child was under observation, and then the cyanosis was most marked. The lips and mucous membranes,

up to the end of the first month of life, were distinctly bluish; the extremities mottled and blue, were with difficulty kept warm. These appearances gradually lessened; the child throve, and as the circumstances of the parents had improved, received more care than had fallen to the lot of its predecessors. When I last saw it (at four months) it was free from all duskiness of its mucous membranes, well nourished, and fairly intelligent-looking.

As difference of opinion still exists in some quarters as to the possibility of a murmur being produced by patency of the foramen ovale, I append a case which has been published by my friend and colleague, Dr. Mackey, of this town, who, as a student of the hospital, had observed with me the cases narrated above, and had heard from time to time my clinical remarks thereon. I had the opportunity of attending the *post-mortem* examination in this case, and of verifying the absence of any condition in the heart or great vessels capable of producing a murmur except the patent foramen ovale.

"A female child, five months old, was brought as an out-patient to the Children's Hospital on October 23rd. Its face and extremities were of dusky colour; it had cough, hurried breathing, and much palpitation, and a distinct murmur with

the first sound over the heart. I did not ascertain whether this was propagated or not up the great vessels. The child had been dark-coloured from birth, and had had convulsions at intervals. It did not seem colder than others. The mother was not strong. She had suffered during pregnancy from palpitation and faintings, and at the seventh month had fallen down stairs. The child was born at the full time. She had two other children, healthy; and she had no cardiac disease. The child was ordered an expectorant and stimulant mixture, and I saw it once more and verified the presence of murmur. A few days afterwards it died in convulsions. On the following day I made a *post-mortem* examination, at which Dr. Foster was present. The fontanelle was not closed. The abdomen was distended. The chest was markedly cone-shaped, the apex being at the neck. The surface was not so dark as during life. On opening the thorax, the lungs were in part pink-coloured and healthy; in part dark and collapsed. The pericardium contained more than the usual quantity of serum. The heart was larger than normal, but in its ordinary position. The right cavities were larger than the left, and contained clots; the left were empty. The walls of the right ventricle were somewhat hypertrophied. The aortic and pulmonic valves were healthy. We examined carefully the orifice of the pulmonary

artery, and there was no contraction about it; on the contrary, it was, if anything, larger than usual: it was so far larger than the orifice of the aorta that it admitted my little finger readily to the second joint, whilst the aorta admitted the same only to the first joint. The foramen ovale was patent, admitting a goosequill above the edge of its imperfect valve. The Eustachian valve was well developed. The liver was large."(6)

(6) *British Medical Journal*, vol 2, p. 666. 1871.

III.

ON THE USE OF ETHER IN THE TREATMENT OF PHTHISIS.

Modern research is daily making it more certain that, under the term Phthisis, we have hitherto grouped together a number of pathological conditions which differ widely in their finer features. But, although the microscope and the scalpel have, for some time past, been busy in defining the distinctive characters of these different pathological conditions, the work is as yet so far unfinished, that, for convenience sake, we still include under the term Phthisis many cases which the more accurate pathology and the more exact diagnosis of the future will enable us to differentiate. In this wide sense I use the term Phthisis in this paper; and I have the less scruple in so using it, because I believe the diseases comprehended under this term, have, as one of their earliest conditions—almost, I might say, as their essential primary phenomenon—a perverted nutrition, from which, as the *fons et origo mali*, all the later phenomena proceed. Errors of nutrition form the starting

point in so many diseases, that it is not improbable we shall have to investigate the earlier phases of these nutritive disorders with more penetrating scrutiny, before we discover the subtle causes which determine the varied results of inflammatory cell-growth—to cite an extreme instance, to learn why in one case cancer and in another tubercle is produced.

The general acceptance of the doctrines of cell-pathology has made us too solidistic in our views. We have all been too busy watching the results of tissue irritation, and observing the changes in tissue elements produced by irritation, to give much attention to anything else. However independent of the blood the origin of the inflammatory process may be, the tissues can only grow and their cells proliferate in proportion to the quantity and quality of the blood supply. The individual cells are not each independent centres of action, they must depend on the pabulum which the nervous and circulatory systems permit them to receive. The influence of the quality of this pabulum in modifying the products of tissue-change has been too generally overlooked. The products have been described with the greatest accuracy, but the conditions which favour the occurrence of this product or another, and which predispose certain tissues rather than others to inflammatory cell growth, have been neglected.

It is surely reasonable to suppose that the chemical character of the new growth, will be in close relation to the chemical character of the blood supply, and that the same relation between blood and tissue will predispose certain organs to the inflammatory process. In the case of those lung changes which we group together under the term Phthisis, we should expect to find a certain chemical similarity between the diseased products in the lung and the constitution of the blood. Analysis after analysis of tubercle has been made, with the constant result of showing that it is a highly albuminous product. These analyses have not been confined to what we now call true tubercle, but have embraced the various forms of tubercle described by the older authors, many of which we now know to be simple inflammatory products derived from the tissue elements of irritated lung. The analyses are on this account all the more valuable for my purpose, inasmuch as they show that the inflammatory process in lung tissue demands a large quantity of albuminous material for the development of the products which characterise pulmonary consumption. Now, it has long been a matter of remark, that the digestive changes which precede the occurrence of Phthisis are distinguished by a special inability to digest fatty food, while the digestive power over albuminous matter is unimpaired. The consequence is ob-

vious: the albumen is assimilated in excess, and albuminous tissues, such as compose the lungs, find plenty of fuel in the blood to supply their inflammatory growth, whenever the slightest irritation excites cell proliferation. That digestive changes which favour the assimilation of a relative, or actual excess of albumen, should, through a highly albuminous blood, predispose to inflammatory growth in albuminous tissues and aid the formation of albuminous growths, appears to me a rational hypothesis, and one pregnant with suggestions for the successful treatment of pulmonary consumption and allied conditions of mal-assimilation.

The disorders of digestion associated with pulmonary consumption have long been known to the profession, but it is only of late years that they have attracted special attention. Numerous independent inquiries have all ended by pointing to the difficulty of assimilating fat as the constant characteristic of the dyspepsia of Phthisis; and statistical observations tell that, in at least 75 per cent. of consumptive patients, this defective assimilation occurs. Adding this fact to others, such as the early and rapid disappearance of the fat stored in the tissues, the development of the inability to digest fat, antecedent to the local lesions, and the marked improvement observed

in patients when the digestion of fatty matter is restored, we have, I think, evidence strong enough, in the absence of any more precise indications, to demand that our first efforts should be directed to improve this state of defective assimilating power. Some years ago, Mr. Jonathan Hutchinson, writing on this subject, said, "Exceptions undoubtedly do occur, but, as a general rule, it might probably be safely laid down that the severity of the tubercular dyscrasia is measured by the difficulty with which cod-liver oil is borne. The need for that remedy is mostly in exactly inverse ratio to the facility with which it is digested."[1]

The greatest step made for many years in the treatment of consumptive patients was the introduction of cod-liver oil; and Dr. Hughes Bennett, to whom we owe this great boon, expressly states "that the great obstacles the practitioner has to contend with are the dyspeptic symptoms."[2] In cod-liver oil was found a form of fat which could, in many cases, be digested even by the impaired organs of consumptives; but the next step, viz., the search after some means of augmenting the secretions which are specially devoted to the digestion of fatty matters has been hitherto neg-

(1) "On the form of Dyspepsia which often precedes and attends Phthisis."—*Medical Times and Gazette*, vol. i, 1855, p. 383.

(2) *The Pathology and Treatment of Pulmonary Consumption*, 2nd edition, p. 114.

lected or unsuccessful. The difficulty with which the remedy is borne by many consumptives, arises in part from the nauseous flavour of the oil, but more especially from the defective assimilation of all fatty food, which characterises so large a proportion of these patients. The abhorrence of all fat, which so many consumptives express, makes it of very little use to order the remedy without first taking means to insure, not only its tolerance, but what is much more important, its digestion. This has been generally insisted on in theory, but I am afraid, nevertheless, generally neglected in practice; and cod-liver oil has been prescribed for years, and is still being prescribed, without due reference to the power of the patients to assimilate it. To pour oil into a patient's stomach, without at the same time taking measures to insure its digestion, has always appeared to me a crude kind of therapeutics. Its simplicity has, it would seem, recommended it to the routinist; for there is probably no single remedy used in any disease with so little preliminary inquiry into the conditions favourable to its action. The attention of prescribers has been too much directed to masking the nauseous flavour—to tricking the stomach into accepting the drug, and too little to the treatment of that morbid condition of the digestive fluids, of which the horror of fatty food is simply the outward expression. In Phthisis,

more than in any other disease, we meet with this inability to digest fatty articles of diet as a symptom demanding our attention, and often persisting in spite of our most elaborate therapeutical formulæ. A sense of the unsatisfactory resources at my command wherewith to combat this symptom long ago impressed itself on me, and made me dissatisfied with the formal therapeutics which prescribes cod-liver oil as the routine remedy for all consumptives, without any sufficient preliminary inquiry into their ability to digest it.

The reform of this practice appeared to require a careful search into our knowledge of the digestion of fat, and a further search for some agent capable of influencing the secretions of those glands which are specially devoted to assimilating fatty food. For a long time it has been known that the digestion of fatty food is performed in the upper part of the small intestines, and that the secretions of the pancreas and duodenal glands, which are poured forth to meet the food as it leaves the stomach, have a special transforming power over the fatty constituents of our diet. To these secretions therefore, we must look for assistance in the treatment of phthisical patients by cod-liver oil, for on the action of these glands must the assimilation of the remedy depend. And such healthy action can scarcely be supposed to co-exist with the inability to digest

fat, which is so frequent in the disease under notice. As Bernard has shown, the chief of these glands (the pancreas) is most sensitive to nervous influences, ceasing to form a healthy secretion under very slight irritation, and even under emotional influences. This fact, taken together with our knowledge of the peculiar character of the dyspepsia of Phthisis, justifies the assumption that the pancreatic secretion is disturbed. Not long since, Dr. Dobell, holding these views, made some experiments with pancreatic emulsions of fat and pancreatine, which supported this interpretation of the phenomena, and showed that fat when emulsified is more readily digested than in any other form. In this paper I have, however, to propose a mode of treatment which should, I think, in all cases precede the use of such remedies as pancreatic emulsion, because it aims at remedying the disorder, not by artificially supplying the defective secretion, but by stimulating the glands to a renewal of their healthy action. Instead of throwing into the system a substance which may be yet formed in physiological quantities, it endeavours to promote the normal flow of pancreatic juice.

The drug which gives us this power is *Ether*, and I now propose to lay before the meeting[3]

(3) This paper was read at the Meeting of the British Medical Association at Oxford in 1868.

the evidence of its physiological action, and afterwards to speak of its therapeutical effects. I had long sought, and sought in vain, for some means of acting on the pancreas in the treatment of Phthisis, till one day, two or three years ago, when reading Claude Bernard's elaborate investigations of the properties of pancreatic juice, I found the long looked for clue; stated, indeed, so simply, and brought out in successive observations and experiments so clearly, that it has ever since been a matter of surprise to me that no previous reader should have seen the importance of the facts to practical medicine. In the following extracts, I lay before you the evidence of our greatest living physiologist on the mode of stimulating the secretions of the pancreas. The first passage which arrested my attention was the following, which occurs in Bernard's *Leçons de Physiologie Expérimentale appliquée à la Médecine*, tome ii, p. 226. "The nervous influences which directly excite the secretion of the pancreas are much more difficult to determine than those which act on the salivary glands. In a dog, with a tube fixed in the pancreatic duct, I have galvanised the solar ganglion of the great sympathetic without a result, clearly showing any modification of this secretion. *But I have seen ether introduced into the stomach determine soon afterwards a considerable flow of pancreatic juice.*" This statement was

so clear and unmistakable, that I at once proceeded to search in Bernard's other works for its confirmation. From his *Leçons sur les Effets des Substances Toxiques et Médicamenteuses*, Leçon 29, p. 426, I extract the following: "I would remind you only of the influence of ether on the intestinal secretions. Ether is reduced to a vapour when introduced into the stomach, and may rupture it.[4] When, however, this accident does not occur, then may be noticed a vascular congestion of all the digestive tracts, but this congestion never attains to inflammation. At the same time the secretions are rendered active and the rapidity of absorption is notably augmented. These last effects suffice to establish the non-inflammatory character of the congestion of the mucous membrane, for it is known that on an inflamed surface the secretions are dried up and absorption becomes more difficult. . . . The turgescence of the mucous membrane of the intestine, as well as the liquids secreted, present the same characters when produced by ether as when they are caused by their normal excitant—food. In comparing the pancreas of an animal, opened fasting, with that of an animal opened during the process of digestion, a great difference of colour, due to the difference of vascularity, is observed. After

(4) This can only occur in those lower animals which have not the power of regurgitation.

the administration of ether the pancreas becomes red and turgescent, as it is during digestion, and its secretory function is proportionally increased by the afflux of blood. I have accordingly been able to avail myself of this procedure to obtain pancreatic juice. Normally, this fluid issues by drops more or less frequent, but when a little ether is given to an animal having a pancreatic fistula, the discharge is seen to become more abundant, without the secreted liquid having lost any of its physico-chemical characters." Demonstrating these facts to his class, the Professor continues: "Here is a rabbit upon which we will show you this increased secretion under the influence of ether. By the procedure already described we lay bare the pancreatic duct, and into it we introduce a small silver tube; you perceive the intestines of the animal red, very vascular, and containing ether in vapour; the animal has received into its stomach 5 cc., which were immediately vaporised and diffused in the stomach and intestines. The animal is lying on its left side, and is in a state of complete anæsthesia: this effect is very easily obtained by giving ether by the stomach in the case of rabbits, but with more difficulty in the case of dogs, as you know. The tube which we have introduced into the pancreatic duct evidently gives passage to a very active flow of pancreatic juice, while in the

ordinary conditions the liquid flows drop by drop with much less frequency. I would remind you again that when the pancreatic juice flows in greater abundance it becomes more liquid and more watery, a condition which is not a consequence of the administration of ether, but one which occurs in the physiological state. Ether we have seen increases the absorption of fatty matters, which it reduces to a state of very fine division, and brings in contact with a more abundant secretion of pancreatic juice. In this way it produces a very marked white injection of the lacteals. Ether enables us to demonstrate on the living animal that the pancreatic juice alone emulsifies fat: a demonstration difficult in the ordinary conditions of digestion, on account of the time it requires and the sources of error to which the pathological state caused by the operation may give rise. If two rabbits are taken as young as possible, and then the pancreatic duct tied in one of them, and into the stomachs of both a solution of fat in ether be injected, there will be seen, on afterwards opening these animals together, that in the rabbit whose pancreatic duct was tied the lacteals contain only transparent lymph, whilst in the other rabbit they are filled with a milky lymph."

In another place (p. 422) summing up the results of his experiments, and alluding first to the

effects of ether in promoting absorption of fatty matters, he says:—"Finally, in our experiments, the injection of the lacteals is due to the abundance of the pancreatic secretion, and to the extremely fine state of division of the fatty matters which occurs in the intestine. It is due, also, to *the absorbing power having been augmented by the ether.* This activity of absorption has been observed in the case of several poisons. We have already mentioned that the combination of ether with poisonous doses of strychnia and nicotine produce death more rapidly on the animals on which we experimented. The action of ether introduced into the digestive organs is, therefore:—1. To stimulate all the intestinal surface. 2. To stimulate the secretions which are poured into the intestine. 3. To modify and decidedly increase the absorbing power."

If necessary, I might multiply these extracts still more; but enough has, I think, already been quoted from the works of our greatest modern experimental physiologist to teach us the physiological action of ether. This action is twofold:—1. It stimulates the pancreas and glands of the duodenum to pour out their secretions freely, and 2, at the same time, it facilitates the absorption of those very substances which these secretions are designed to digest. In other words, ether not only increases the secretions required to digest

fats, but promotes the absorption of these fats when digested. Can any physiological action be more clear, or any experimental proof be more satisfactory for the end we have in view?

I now come to the second part of my task; namely, the therapeutical application of the foregoing facts. Having found, as I considered, sufficient evidence of the special action of ether on the pancreas, I determined to test the value of the discovery by administering ether in all cases characterised by inability to digest fat. In the out-patient departments of our hospitals, many such cases occur, which are generally classed as dyspepsia. Many cases of neuralgia are also to be met with in which a most decided improvement follows the increased power of absorbing fatty food. To both classes of patients I gave ether, sometimes in combination with cod-liver oil, sometimes alone before meals. The results were most satisfactory; the oil was digested more easily, and the nutrition of the patients greatly improved. Of these cases I shall speak more fully on a future occasion. I next ordered ether in mixture to all my phthisical patients at the General Dispensary, selecting the Dispensary for my investigations, rather than the Queen's Hospital, because all the patients were out-patients, and consequently exposed to no new and favourable conditions while

the experiment was being tried. The patients being also of a somewhat better class than those attending the hospital, were not, as is so often the case in our large towns, struggling to combat a mortal disease while suffering from the effects of disease's chief ally—improper and insufficient food. For some months I pursued the treatment, seeing a very large number of consumptive cases, to all of whom ether either alone or in conjunction with cod-liver oil was administered; and the good effects were so decided, that I determined to investigate the subject more methodically, and to keep a careful record of each case. The good results observed in this preliminary inquiry were a return of the power of taking oil and fatty food, which had been previously distasteful or had even excited sickness: increased appetite, improved general nutrition and increase of weight, diminution of cough and expectoration, and cessation of night sweats. The general impression produced in my mind was so favourable, that I began my more exact inquiries with much confidence. In my first cases the ether was given as follows:

℞ Potassæ Bicarb. ʒiss — ʒij.
Acidi Hydrocyan dil. min. xij — xvj.
Spt. Etheris ʒiss — ʒiij.
Aq. ad ℥viij. Misce.
ʒj ter in die sumat.

This mixture I still use extensively, and find it

one of the best, if not the best method of giving ether. The ether was afterwards added to cod-liver oil, the *Æther Purus* of the *Pharmacopœia* being used, about ten minims (9.259 min.) being added to every two drachms of oil at first, afterwards fifteen and twenty minims being occasionally given in the same quantity of oil.[5] In some cases the ether was administered in water alone, and taken a short time before the oil. The effects were similar in all cases. In hospital practice, for convenience sake, and on account of its power of masking the unpleasant properties of the oil, I now generally give ether mixed with cod-liver oil, in the proportions mentioned, while in private practice I prefer the use of the mixture mentioned above, or the administration of the spirits of ether (min. x to xxx) in water, a few minutes before or after the oil is taken. For private patients the etherised oil may be flavoured with oil of lemon and glycerine. A still pleasanter preparation is an etherised emulsion of cod-liver oil, which Messrs. Southall of Birmingham, have prepared for me.

In my second and systematic inquiry, I treated fifty cases taken as they presented themselves at the Dispensary, each patient being carefully

(5) Mr. Barnes, Trevor Terrace, Knightsbridge; and Mr. P. Möller, Oxford Street, London; and Messrs. Southall, Birmingham, have prepared for me etherised cod-liver oil of excellent quality.

examined on admission, and at least once every fifteen days during treatment. A brief record was kept of the progress of each case; but, from former experience, I determined in this inquiry not to accept the statements of the patients themselves as evidence of improvement, but only to consider those patients better in whom the result of a physical exploration of the chest, in addition to *decided increase* in weight, confirmed the statements made. I therefore weighed every patient once a week. As it would be impossible to give a record of each case in a communication such as the present, I must confine myself to a brief statement of the results obtained in the fifty cases.

Sixteen were admitted in the first stage of the disease; nineteen in the second; and fifteen in the third stage.

Of the sixteen in the first stage, seven improved in general symptoms and in physical signs, gaining on an average $7\frac{1}{2}$ lbs. each; five remained stationary, all gaining weight slightly; and only four became worse.

Of the nineteen cases in the second stage, six improved in all respects, gaining, on an average, about 8 lbs. each, two cases gaining $14\frac{1}{2}$ lbs. and 10 lbs. respectively; six remained stationary; and seven became worse.

Of the fifteen cases in the third stage, seven improved, gaining, on an average, about 5 lbs.

each; five remained stationary; and three became worse.

Of the total fifty cases, twenty improved, sixteen remained stationary, and fourteen became worse.

Six of the cases treated, in no instance for a less time than three months, and observed over a period of many months, retained their weight; were troubled by no bad symptoms, and maintained the improvement in their physical signs. Only one of these was observed over a less period than fifteen months, three were under observation over two years, and two over eighteen months. These cases may be considered, I think, examples of the arrest of the disease; for the patients have returned to their ordinary avocations, and are able to earn their livelihood untroubled by their former symptoms. The only other remedies used in these cases were croton oil ointment, as a counter-irritant, Dover's powder occasionally to relieve the cough, and, in a few cases, a little linctus morphiæ..

As cod-liver oil was administered as well as ether, the objection may very naturally be made that the improvement was due to the use of the oil. For my own part this objection has not much value, for I had long used oil largely without obtaining any results at all equal to those stated. In order, however, to meet this objection, the

ether was stopped in ten instances, and the oil continued; six times there was a fall in weight during the following three weeks. In one case the patient, who had taken ether for some two months, continued to gain weight. In the three other cases the gain ceased, but was again observed in these patients, as it was in all, when ether was again administered.

These results are not only instructive, as showing the effect of ether in increasing weight, but also as showing that the digestion of fatty food is restored to its normal state, in many cases, after a long course of ether. In the cases which have done best under treatment, that is, the cases which I think we may consider instances of arrested Phthisis, the power of taking fats, which was at one time much impaired, has returned, and the gain in weight has been maintained over a period of many months. I may here also add that in all the fifty cases there had been marked wasting before my treatment was adopted, in at least half the cases, cod-liver oil notwithstanding. Many of my patients have over and over again expressed their preference for the etherised oil; and many who have been unable to retain ordinary oil when taken, have been able to use etherised oil, not only without disagreeable symptoms, but almost with pleasure: "to drink it from the bottle," as some have expressed it. Not many

days since, in visiting my patients in the General Hospital, I noticed that the shirt of a strumous child, for whom I had ordered cod-liver oil on admission a few days previously, was badly stained with the remedy. I inquired if the oil had been spilled, but learned from the nurse's reply that the child always rejected a greater part of the oil. This had not been mentioned to me before as the nurse thought it well to persevere with the drug. The ordinary oil was changed for the same dose of etherised oil, and with the best effects. The oil was no longer rejected, the very first dose was retained, day after day the child's appearance improved, and his appetite increased. Such cases constantly occur among my out-patients, and when the etherised oil does not at once agree with the stomach, a few doses of a mixture containing ether will almost invariably give the patient the power of taking the oil without any feeling of discomfort.

In advocating a new method of treatment, it is satisfactory to be able to appeal to confirmatory evidence. It gives me great pleasure, in the present instance, to be able to call as a witness in behalf of my views, Dr. E. L. Fox, of Clifton, to whom I mentioned the advantage of prescribing ether in Phthisis in April, 1867. I had the benefit of discussing the subject with Dr. Fox at that time; and he has since been so good as to

send me the results which he obtained. In the early part of this year (1868) he wrote to me as follows: "I fear I have kept no precise records of the oil and ether question; but since you mentioned it to me, I have used it very fairly. I give it in two sets of cases: 1, those who are tired of taking cod-liver oil; and, 2, those who have been taking it without gaining weight. In the first cases I find it useful, as the ether, half an hour after the oil, seems to settle the stomach; and, I believe, induces the digestion of the oil. The nausea felt by so many patients is, I think, merely evidence of the oil not being digested. In one hospital case, the man took oil for fourteen days without any benefit; indeed, lost flesh. I continued the same dose of oil, and added the ether, and he began to gain three pounds a-week, and went on at the same ratio as long as he was in the wards. I think I have given it to about thirty patients since our talk in April, mostly in private cases of chronic phthisis, and in one case of great debility, in which I believed the pancreatic secretion was too little. In this case the young lady had a great horror of all fat things, but took the oil with ether after it fairly well, and gained on it. It seems a plan generally liked by patients, and I am convinced of its utility." I need hardly say how gratified I am to have the support of so able an observer.

I may here allude to the confirmatory evidence of the therapeutical action of ether in promoting the assimilation of fat, contained in Dr. Ramskill's important observations on the use of olive oil as a remedy. From many practitioners I have received strong assurances of the advantages of the combination I have recommended. I will only quote the words of one whose high position as a physiologist and reputation as a clinical observer give special value to his words. I refer to Professor Mapother, of Dublin, who writes as follows:— "Since I had the advantage of hearing your paper at Oxford I have given etherised cod-liver oil in about fifteen cases, chiefly those of strumous and rheumatic diseases of bone. It is far easier to take than the oil alone: the ether acts as a good stimulant, and I think it fattens the patient most surely. Your views as to the increased pancreatic flow and improved emulsification of fat are sound." I can strongly confirm Dr. Mapother's opinion of the value of ether in cases of strumous bone disease. I have had in my own practice some striking examples of the good effects of ether when given in mixture or with cod-liver oil, in arresting the progress of the caries, diminishing the discharge of pus, and improving the general nutrition. I might quote the testimony of others who have used the ether at my suggestion, were it necessary.

I would remark that I have used ether in the treatment of Phthisis, not hoping to find a specific, for we have in these days ceased to search after specifics for disease, but simply expecting to discover a rational means of combating one of the most serious symptoms of the malady, and restoring to its healthy state that perverted nutrition which forms so characteristic a feature in the great majority of consumptive cases. The peculiar characteristic of this disorder of the digestive organs has been dwelt on, and the facts are, I think, sufficiently strong to show that in ether we have the best remedy for renewing the power of assimilating fat, whenever that power is diminished or lost.

Such, briefly told, are the results of my inquiry. I submit them to this meeting, in the hope that others may assist me in testing the truth of the facts advanced. Throughout, I have endeavoured to investigate the question with scrupulous care; the drug was selected only after a lengthened search, and when the evidence of its physiological action appeared to me conclusive. Starting from physiological facts, I have used the more simple science, physiology, to assist me in adding a new fact to the more complex subject, therapeutics; for it is in this way that I believe medicine, a compound of sciences, can be best advanced.

Before a drug can be scientifically applied to the relief of disease, the modifications which it produces on healthy function must be known. When the knowledge is gained we are in a position to apply it to those variations of function which constitute the pathological state.

In the selection of cases for experiment, I have endeavoured to eliminate, as far as possible, all sources of fallacy, by selecting patients in the same condition of life, exposed to very much the same influences; and I have refused to accept any statement as regards improvement, which has not been confirmed by the best test of all, in my opinion, the improvement of general nutrition. The patients, also, were, as far as could be determined, suffering from the same pathological conditions; they were all cases which may be termed chronic phthisis. In short, in all respects I have endeavoured to shut out from my inquiry all the accessory circumstances which might be sources of error; and I trust I have in this way avoided mistaking mere coincidence for the relation of cause and effect. The difficulties of avoiding all such sources of fallacy in a therapeutical inquiry are immense, hence the slow progress which this part of medical science makes towards scientific form. If, in the present instance, my inquiry has been conducted with sufficient care to make it appear that I have indicated a new

direction of fruitful research, my object will be accomplished. I leave it to the experience of the profession to decide on the value of ether in combating one of the most subtle and certain approaches of our great enemy—death.

IV.

DIGITALIS IN HEART DISEASE.

Gentlemen,

Nearly one hundred years have elapsed since Dr. Withering, a former physician of this hospital, published his celebrated *Account of the Foxglove*. In that work the curative powers of digitalis in certain forms of dropsy, and its remarkable influence in diminishing the frequency of the heart's action, were proved by the investigations of a most able observer. A century's experience would appear to any one unacquainted with the slow evolution of scientific therapeutics, more than enough to have completed the inquiry begun by our illustrious predecessor, and to have determined the true position of the drug in relation to that organ, on which the first investigator noticed its effects.[1] Not so; there are still to be found in the text-books of the profession the most

(1) In the ninth inference at the end of his essay, p. 192, Withering thus expresses his opinion of the action of digitalis on the heart: "That it has a power over the motion of the heart, to a degree yet unobserved in any other medicine, and that this power may be converted to salutary ends."

diverse opinions as to its action, and the vaguest directions for its use. The drug which long ago earned the title of "the opium of the heart," is even now prescribed in the most opposite conditions, for the most contradictory reasons, and, as might be expected, with the most varied results.

The absence of clearly defined principles as to the forms of heart disease in which its good effects may or may not be expected, has led to all this confusion. Digitalis has been and is still ordered, when no exact diagnosis has been made; and this haphazard practice has necessarily led to widely different estimates of its value.

During the last few years my sphygmographic observations have led me to devote much time and thought to the study of heart disease, and the action of digitalis has consequently been a matter of daily observation and comment. It is impossible to go on using a remedy day after day in special forms of disease without, drawing some conclusions as to its effects, and framing some rules for its administration. To-day I propose to sketch the line of thought which has guided me in the use of this drug. The argument may, perhaps, possess little novelty, and the conclusions may not be new, but at all events I hope they may prove useful to you as they have to me.

In the beginning of my inquiry into the action of digitalis, finding no authority to guide me in

the selection of cases for its use, I made a series of observations on the effects produced by it when administered to healthy persons, and to patients free from heart mischief. Giving it in moderate doses, one to three drachms of the infusion three times a day, I obtained results agreeing with those of other observers. The pulse traces showed—

1. A diminution in the frequency of the heart's beats.
2. An increase in the force of each beat.
3. An increase in the arterial tension.

In cases of irregular action of the heart associated with certain valvular lesions, there was observed what might be called a fourth effect, viz.,

4. The co-ordinating influence of the drug in restoring the regularity of the heart's movements.

That these effects are produced by the action of digitalis through the sympathetic nerve on the heart and smaller blood-vessels, there is, I think, almost complete evidence. Whether the action is first on the heart or on the blood-vessels is another question, but one not primarily important for our present purpose, since I now only propose to consider what indications we can gather as to the use of the drug from its admitted effects on the heart. In considering these effects, we must regard the heart mainly from a mechanical

point of view; as a pump furnished with stop-valves to direct the stream of blood aright. When these stop-valves get out of order, there is no opportunity for repair, for there is no possibility of complete rest. The valve lesion, since it cannot be remedied by direct repair, is compensated for by alterations in the size of the heart cavities, and in the power of the heart muscle. By these means the defect of the valve is reduced to a minimum as regards its injurious action on the circulation. The pump still does its work, not perfectly, it is true, but often so nearly perfectly that with judicious aid it may perform its functions for years. It is the mechanism of the compensation in each form of valve lesion which must be mastered, before correct ideas of treatment can be formed, or the true position of any drug as a remedy in heart cases can be defined. I propose to consider the several forms of valvular disease in succession, and I shall first take aortic insufficiency.

I.

Aortic insufficiency.—In this form of valvular mischief we have a morbid change which acts only during the diastole of the ventricle. From the moment the contraction of the aorta on its contents takes place, and forces down the imperfect sigmoid valves, the blood pressure in the

aorta drives a stream of blood between the insufficient valves into the dilating left ventricle. This regurgitation continues till the pressure of blood in the ventricle becomes equal to that in the aorta, that is to say, till the ventricle is filled ready for its next contraction. This period, during which the regurgitation acts, is very nearly half the period of each cardiac revolution. The results of this reflux are well known; the circulation is impeded, the balance between the contents of the arteries and the veins is disturbed by the loss which the arterial side suffers during each diastole, and a state of low arterial tension is produced. If the insufficiency be suddenly created, a rapid development of pulmonary engorgement and a general venous stasis occur, which may end in death. In a case of this kind admitted under my care last year, in which the diagnosis of ruptured aortic valve was afterwards verified, the accident was followed by insensibility, general cyanosis, and total absence of the radial pulse; it was only by the most untiring care in the administration of stimulants, and the judicious abstraction of some twenty ounces of blood, that Dr. Welch (then house physician) was enabled to restore the circulation.

Aortic insufficiency acts on the circulation in a purely mechanical way. The aortic stop-valves are imperfect, and consequently the whole period

of their action is a period of detriment to the efficiency of the circulation. To compensate for this, certain changes take place; the left ventricle dilates and hypertrophies, and the action of the heart becomes more rapid. More blood is thrown into the aorta, and less time is allowed for its regurgitation. What action would digitalis have in this case? It would augment the ill-effects of the insufficiency by slowing the action of the heart. The diminution in the frequency of the heart's beats under digitalis, always means an increase of the period of the dilatation of the ventricles. Pulse traces readily show this. It is during this very period that aortic insufficiency produces its ill effects, and it is, consequently, not hard to understand why digitalis acts injuriously. The low arterial tension may require increasing, the heart's strength may want reinforcing, but these two indications cannot be fulfilled by any remedy which, like digitalis, adds to the prime evil. The more forcibly the heart beats, and the greater the arterial tension, the greater, *cæteris paribus*, will be the reflux through the imperfect valves; and while the valvular lesion remains a constant quantity, digitalis will multiply its effects by increasing the period of its action.

But digitalis has been known to do good in aortic insufficiency. I admit this. There is one condition which warrants its use—that of over-

compensation. This is marked by violent action of the heart, bounding vibratory arteries visible all over the body, almost constant headache, flushed face, noises in the ear, occasional epistaxis, urine normal in quantity and free from albumen, &c. In these cases a few doses will do good. The action must, however, be watched, and the moment the pulse falls the remedy must be stopped; if not, syncope and other alarming symptoms occur.

A few weeks ago two cases in the wards served to illustrate these remarks. The first, you may remember, was that of a finely-built muscular man, æt. 35, with well-marked aortic insufficiency of some twelve months' standing. His general symptoms, as well as the characters of his pulse and impulse, indicated that the hypertrophy of the left ventricle was insufficient to render the compensation perfect. So small a quantity as three drachms of infusion of digitalis, given in half-drachm doses, aggravated all his troubles. The pulse became slower, but feebler in its tension, the dyspnœa increased, the urinary secretion was diminished, and a feeling of faintness and sinking was complained of, which gradually became almost intolerable. The continuance and repetition of the remedy increased these symptoms, the administration of brandy relieved them.

The second patient was a girl of 16, who had aortic insufficiency of some eighteen months' dura-

tion. In her case the very rapid incompressible pulse, the violent impulse, the flushed face, the almost constant frontal headache, the comparative absence of dyspnœa, and the free secretion of urine—all told of an hypertrophy of the left ventricle, sufficient, and more than sufficient, to compensate for the valvular lesion. Half-drachm and afterwards drachm doses of the infusion were given with the best effects. The headache was relieved, the pulse fell in frequency, the violent heart action was lessened; but even in this case the remedy could not be borne long. On the fourth day sickness and faintness and diminished flow of urine warned us to withdraw it. It is on this account that I generally prefer hydrocyanic acid, caffeine, and sometimes aconite, in these cases of over-compensation. They act equally well, and are less liable to cause unpleasant symptoms than digitalis. But the drug which gives the most rapid relief to the headache is nitrite of amyl. The inhalation of the vapour of a few drops of the nitrite on cotton wool cures the headache, lowers the arterial tension, and quiets the excited heart.

II.

Mitral stenosis.—Narrowing of the left auriculo-ventricular orifice by adhesion and contraction of the curtains of the mitral valve, constitutes a

purely mechanical obstacle to the circulation, which acts, during the same period of each cardiac revolution as aortic insufficiency. In this case, however, the filling of the ventricle is impeded by the narrowness of the passage through which the blood has to enter. While aortic insufficiency renders the ventricle over full, mitral stenosis keeps it too empty; yet both tend to drain the arterial side of the circulation: the former by allowing the blood to flow back, the latter by opposing its advance. Now, the period of ventricular dilatation being that during which the blood flows from the auricle to the ventricle, when there is marked narrowing of the channel of communication, the impediment can be compensated for in two ways. The propelling power must be augmented to drive the blood at a greater speed, or the time for its passage must be lengthened.

When the narrowing is not very considerable, and the demands on the circulation by exercise are limited, a very perfect compensation is effected by the hypertrophy and dilatation of the left auricle and the right ventricle. By these means the current of blood through the lungs is made more rapid, the tension in the auricle is increased, and the velocity of the current filling the left ventricle is so multiplied, that the balance of the circulation is fairly maintained, in some cases for

years. Only the other day some of you saw a patient suffering from this form of valve disease, who came to seek me in the out-patient department, after an absence of nearly nine years. She was somewhat worse than she had been formerly, it is true, but she still enjoyed a comparatively comfortable existence, and was still capable she said of doing all her domestic work without distress. Since she had been under my care she had married, but like most women suffering from chronic valve disease before marriage, she had never conceived.

When, however, the narrowing is considerable, or some unusual strain has disturbed the unstable equilibrium on which the health of these patients depends, then the compensation which the auricle and ventricle offer fails. The rapid, irregular pulse tells of the varying quantities on which the ventricle contracts; sometimes the interval between the ventricular contractions is so short, and the ventricular charge is so small, that the systolic wave does not reach the wrist. Under these circumstances the second indication must be followed; the auricle must have more time to fill the ventricle. This, to say nothing of the increased power given to the cardiac muscle, is exactly what digitalis affords. By slowing the action of the heart, the period of time during which the blood from the distended auricle can flow into the ven-

tricle is increased, and as the extra time allows more blood to pass through the narrowed mitral orifice before the final effort of the auricle is made, that final effort is made on a smaller quantity of blood, and is, consequently, more effective; for the smaller the quantity of blood which the auricular muscle has to push before it, the greater will be the velocity given to the current. The ventricle, though contracting less frequently, contracts more effectually. Instead of eighty or ninety irregular contractions a minute, no two succeeding ones equal in force, and some so valueless that they are not perceptible at the wrist, we get some sixty steady equal beats. The pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy recognises its master, and beats a sullen retreat.

Yet even in this form of valvular disease, in which it is of all drugs the most valuable, digitalis has been declared to be inadmissible. It is, however, only in the very last stage of mitral stenosis that the remedy fails, and then because no drug can restore the functions of an organ irrecoverably worn out. You have seen with me many cases which illustrate what I have said, and looking at this form of disease clinically, I think we may recognise three classes of cases. In the first the

pulse is quite regular or nearly so: the arterial tension though low, is not associated with marked pulmonary congestion; and the patients, under favourable conditions, suffer but little. Such was the case referred to above. In these cases digitalis is not often wanted; but whenever the patient complains of more than usual dyspnœa and diminished urine, with increased rapidity of pulse, it is a safe remedy, and one which almost invariably relieves the symptoms. To such cases among the out-patients you see me give ʒj or ʒij of the infusion, combined with perchloride of iron, three times a day. In the second class the pulse is irregular, and all the symptoms are worse. Then digitalis is still more striking in its effects, but to obtain these fully the patient should be kept in bed. In the last class it is generally too late to get good results. The albuminous urine, the pulmonary complications in the shape of oft-recurring hæmorrhage, and the general dropsy, tell that the heart is hopelessly unequal to its task. Occasionally, even in these cases, digitalis will succeed, especially if preceded by the abstraction of a few ounces of blood, so as to relieve the venous distension. The duration of the albuminuria is, in these cases, as it is in all cases of valvular disease, the most important point whereon to found an opinion; the chance of recovery is generally in inverse proportion to the duration of this sign. Two cases in which digitalis

failed me unexpectedly, were examples of mitral stenosis, associated with a similar lesion on the right side of the heart. In the first case the digitalis was powerless all through; in the second it saved life several times, but on a last occasion failed, and then, after death, as had been diagnosed during life, tricuspid narrowing was found.

III.

Mitral insufficiency, like aortic obstruction, differs from the two preceding forms of valvular disease, in the time during which the valve defect comes into play. It is a deficiency of stop-valve action, which occurs only during the systolic period of each cardiac revolution. The valve action, too, differs from that of the sigmoid valves, inasmuch as it does not depend solely on physical conditions, but is the result of muscular energy. The small muscles which are attached by such a wonderful arrangement of their tendons at the edges of the two curtains of the mitral valve, must act normally to insure the perfect function of the valve. The more firmly and the more steadily these papillary muscles contract, the more perfectly will the valvular curtains be brought into apposition, and the smaller will be the defect in the stop-valve action. While, on the other hand, the more irregular and wavering the action of these little muscles, the less close and the less sure will be the apposition

of the valvular curtains, and, consequently, the greater will be the reflux of blood. The stronger the contraction of the ventricular muscle therefore, the more perfectly will the orifice be closed, and regurgitation checked. The curtains of the valve in health so overlap that between them no passage is left, and the approximation [2] of the muscular sides of the orifice, which possibly occurs to some extent with the ventricular systole, may prevent the thin valve from having to bear the whole pressure of the blood during the ventricle's contraction. In disease, the valves no longer meet in such perfect apposition, and in the space between them the blood finds a regurgitant passage. The compensation by which this is met consists in dilatation and hypertrophy of the left auricle and right ventricle, aided by hypertrophy and dilatation of the left ventricle. The last cavity by the form of its increase contains its extra quantity of blood in the upper part of the ventricle, nearer to the aorta, and more out of danger, as it is further removed from the non-approximated edges of the mitral valve. There is less blood at the apex of the distended cavity where it can regurgitate, more at the base where it cannot. To keep up the compensation there must, however, be good steady muscular action, no wavering in the contraction,

(2) Onimus. *Journal de l'Anatomie et de la Physiologie*, Juillet, 1865. Paris.

and no unsteadiness in the action of the papillary muscles. Any extra effort soon disturbs the artificial equilibrium, and confused muscular action follows. It is this which digitalis corrects. In place of a large number of ineffective contractions, it concentrates the power of the ventricle on a smaller number of well-directed, steady beats, each throwing a larger charge of blood into the arteries and so diminishing beat by beat the over-distension of the right heart. The right ventricle so aided, is at the same time aiding, by the more vigorous efforts which the digitalis enables it to make. The symptoms in cases of mitral insufficiency which specially indicate its use are similar to those which it relieves so marvellously in mitral stenosis. The feeble flickering pulse, the congested lungs, the dropsical limbs, the swollen jugular veins and cyanosed lips and face all improve under its use. There are no cases in which the influence of the drug rightly applied appears more magical. In this form of disease all turns on the healthiness of the cardiac muscle; the remedy will do no good, nay, rather will do much harm, if muscular degeneration has occurred. In the cases in which it has disappointed me I have invariably found after death the heart cavities dilated and the muscles altered.

IV.

Aortic obstruction.—In this condition, the ventricle having to drive its contents through a narrowed outlet, must have either more time to do it in or greater propelling power. The compensatory dilatation and hypertrophy insure both. The ventricle contracts on a larger quantity of blood than is normal, and takes longer to drive it into the aorta. The systolic period of each cardiac revolution is lengthened, and the diastolic period is proportionally increased. In this case we have at once the simplest and the most perfect of any of the forms of compensation. Digitalis can do little for such cases, because there is little good to do. As long as the cardiac muscle is well nourished there is no need for the remedy; when the muscle ceases to be healthy digitalis is worse than useless. The heart acts in these cases slowly and forcibly, and the slowing action of digitalis becomes its dangerous action, and more than counterbalances any good effects which might be expected from its influence on the cardiac muscle.

Not long since you saw me order digitalis in a case of pure aortic obstruction. The pulse fell under its use in the course of three days from 62 beats a minute to 50. This diminution in the frequency of the heart's action was associated with no improvement in the symptoms. On the

contrary, the patient, who had been comfortable before its occurrence, now complained of a feeling of faintness and giddiness, and had decidedly increased dyspnœa. The remedy was withdrawn, and all these symptoms disappeared. Some few years ago, a man came under my care for chronic bronchitis, whose pulse beat ordinarily only 27 times a minute. He had no discoverable heart disease, and some years after the heart was found to be healthy. Digitalis was cautiously administered to this patient; the pulse fell to 23 and 24 a minute, and a sense of precordial anxiety and slight attacks of vertigo were produced. On the discontinuance of the drug these symptoms ceased to trouble him, and the pulse returned to its ordinary rate of 27.

It is only when the hypertrophy exceeds the limits of compensation that digitalis is useful; then, by slowing the pulse and steadying the heart action, its good effects are well seen.

The remark may be made that, in practice, we do not meet with the simple valvular lesions I have discussed. I readily admit this truth. Valvular diseases are frequently complex. Aortic insufficiency is often (though not so often, I think, as the systolic murmur would lead us to infer) associated with obstruction; the two forms of mitral disease are frequently conjoined; or

mitral in either of its forms, or double, may coexist with double aortic murmur. But for all these cases the considerations I have advanced are, in my experience, sufficient to guide your practice aright. Both forms of aortic disease are present; we have to treat the one which predominates, and to which the general symptoms are due. This is almost always the regurgitation, and digitalis is hurtful as a rule. In double mitral we have a coincidence of two lesions, both demanding digitalis, and we may give it with the best effects. When mitral regurgitation is developed in the course of a case of aortic regurgitation, we have a rupture of the compensation of the aortic lesion, and digitalis will not help us to remedy it. The rule is in all cases, to treat the predominant valve lesion, and never to expect good from the use of digitalis when the heart muscle is unsound. Guided by the principles I have sketched, you may give it most usefully and most safely. I constantly prescribe it for hospital out-patients from a distance, and continue its use for weeks. I nearly always prescribe the infusion, and test its continued action by the effect on the secretion of urine. As long as the quantity of urine increases or keeps up to the maximum, which the digitalis has produced, the drug is acting beneficially. This rule will always guide you aright. Not long ago, one

of our most eminent authorities on heart disease wrote:—"In cases of mitral valvular disease I believe, however, that digitalis is eminently useful, not by any influence which it exerts over the heart itself, but from its powerful diuretic action."[3] This sentence is full of practical wisdom, but we may now say, I think, that the beneficial effects are not, as Peacock puts it, on account of its diuretic action, but the converse; that the diuretic effect is the result, the outward and visible sign, of the beneficial action. The diuresis indicates a restoration of the normal balance between the contents of the arteries and the veins, an increased arterial tension, and, consequently, a refilling, under normal pressure, of the empty capillaries of the Malpighian bodies. The high-coloured scanty urine, loaded with urates, is replaced by a clear and copious stream, which tells of a steadily beating heart and a firmer pulse.

To some of you the question may occur, how does digitalis, if it act as I have described, produce results lasting long after its administration has ceased? The answer appears to me simple: the continued improvement is a matter of cardiac nutrition. In all valvular defects when the symptoms call for medical treatment, the nutrition of the heart has suffered in some degree. Digitalis,

(3) *On some of the Causes and Effects of Valvular Disease of the Heart.* By Thos. B. Peacock, M.D., F.R.C.P.

prescribed as I have recommended, restores the disturbed balance of the coronary circulation, which has been injuriously affected in common with that of the whole body. With a weak irregular heart action the coronary arteries suffer just as the other arteries of the body, and the heart muscle is badly nourished, not only because its arteries are ill filled, but because its veins are imperfectly emptied. Digitalis restores the normal balance between the contents of arteries and veins. The heart muscle gains strength, the heart beat gains power, and while the renewed vigour lasts the compensation of the valvular lesion is fairly perfect, and our patients have no serious symptoms. So they often go on for months, sometimes for years, till some extra anxiety, some heavy work or some unusual effort overtaxes the imperfect organ, and disturbs the nice adjustment between defective valve on the one hand and altered wall and cavity on the other, which constitute compensation. If the nutrition suffers not too greatly, digitalis may again restore the balance and bring relief once more to the labouring heart.

V.

ON RUPTURE OF THE AORTIC VALVES FROM ACCIDENT.

Gentlemen,

The case of heart disease to which I wish to call your attention to-day is of much interest, as illustrating one of the rarer causes of valvular incompetency. The man H.[1] was admitted some ten days ago suffering from great dyspnœa, palpitation, and œdema of the legs, with the physical signs of aortic insufficiency. His expression was anxious, face pale, carotid pulsations very distinctly visible, cough frequent and troublesome, with little or no expectoration, respirations 48, radial pulse jerky and thrilling, beating 112 times a minute. On further examination the lung percussion-note was found to be healthy, and the breath sounds natural anteriorly, but posteriorly there was dulness at both bases, extending as high as the inferior angle of scapula on the left side and into the axilla as far as the heart dulness. Over the dull

(1) Case reported by Mr. E. S. Warrillow, Clinical Clerk.

area the breath-sounds were indistinct, and there was diminished vocal fremitus on the left side. The area of cardiac dulness was greatly increased, and extended from the second to the seventh intercostal spaces, and from one inch beyond the right edge of the sternum to the dull pulmonic region outside the nipple-line. There was no distinct apex-beat, but a diffused systolic heaving seen in the fifth, sixth, and seventh intercostal spaces, and in the epigastrium. Auscultation gave the following results:—At the base of the heart there were heard two murmurs—viz., a short blowing almost flapping murmur with the first sound, followed by a loud harsh murmur which replaced the normal second sound. The systolic murmur was heard most distinctly at the junction of the second right costal cartilage with the sternum and over the midsternum opposite the third and fourth ribs. It was not propagated with any distinctness, either along the great vessels or towards the apex. The diastolic murmur was heard loudest at midsternum, opposite the third costal cartilage, was distinct at the ensiform cartilage and at the manubrium sterni, and was audible under both clavicles and in the carotids, replacing the second sound; it was not propagated towards the left apex, but was audible down the spine in the interscapular region. At the apex of the heart there was a murmur audi-

ble with the first sound, louder and longer than that heard at the base, and propagated towards the axilla. The diastolic murmur, so loud at the base, was scarcely audible at the left apex, but became more distinct as the stethoscope approached the ensiform cartilage. The abdomen was rather full, especially in the upper part from the enlarged liver, which extended three finger-breadths below the costal arch, and was tender on pressure. There was also evidently some fluid in the abdominal cavity. The urine was scanty, high-coloured, with a copious deposit of urates and about one-quarter of a column of albumen. There was some œdema of the lower limbs.

From these data we concluded that the case was one of insufficiency of the aortic valves, with slight aortic obstruction, and secondary insufficiency of the mitral valve, leading to engorgement of the lungs, liver, and general venous system, and to serous effusion into the pleural cavities and peritoneum. The case was regarded at the time of admission as illustrating defective compensation and the supervention of asystoly.

Our next step was to find out, as far as possible, the circumstances which had led to these troubles. The patient's previous history gave us the clue. He had been originally brought up as a jeweller, but some fourteen years ago he enlisted in the Royal Marines, served in India, China, and through

the Abyssinian expedition, then, having completed his first period, obtained his discharge. Throughout his period of service, as previously, he had enjoyed splendid health; never had syphilis, never had rheumatic fever, and although he drank freely, never suffered from it. After his discharge he returned to Birmingham, and worked for a time at Soho. In February, 1872, he emigrated to America, but, finding no occupation, he enlisted in the American service as a marine. At this time he was in good health, and passed his medical examination as a sound, strong man. His ship was ordered to Valparaiso, and while on the voyage he volunteered to act as cook, and did the baking for the ship's crew (500 men). During the voyage there was some very heavy weather, and one day, on coming up on deck, he was thrown violently forward by a lurch of the ship. He made a great effort to save himself, and partially did so with his right hand by catching hold of the side of the hatchway, but was struck in falling on the left side of the chest. He felt faint and hurt internally, was taken below, and for two days was unable to continue his work. When he resumed it, however, he found the cooking too much for him, and he was obliged to give it up. Shortly afterwards, while on watch one night, and all being quiet, he thought he would lie down on deck.

He was much surprised, however, to find that the perfectly horizontal position made his breath, which had been short since the accident, much worse—so bad, indeed, that he was obliged to get up and walk the remainder of his watch. He mentioned this to his medical officer, who examined him, and on his arrival at Valparaiso invalided him, and sent him back to Norfolk Island, whence he was discharged, and sent home to England. On the homeward voyage he was very ill; his legs and body began to swell, and on landing at Liverpool he was sent to hospital. He recovered sufficiently to return to Birmingham in August last, and from then to the time of admission (November 7) he has been troubled with cough, shortness of breath, and more or less œdema of the limbs.

From this history, which told of no rheumatism, nor other cause of heart mischief, but of perfect health (confirmed by the medical examination for admission to the American service) up to the date of an accident, we could draw but one conclusion —that the aortic insufficiency was primarily due to a rent of one or more of the valve segments produced by the fall, or rather by the effort he made to save himself.

For some days after admission he improved a little, and under the use of morphia, given hypodermically, he had more refreshing sleep than he

had had for weeks, and the dyspnœa became less urgent. Several attacks of syncope, however, occurred; his stomach refused nourishment; and although for two days these syncopal attacks were averted by the free use of liq. ammoniæ, ether, and brandy, he died quite suddenly early one morning from syncope.

The post-mortem examination was made by the Resident Pathologist, Dr. A. H. Carter. On opening the thorax, the lungs were seen to be widely separated by a very large heart. The pericardium was empty, but each pleural cavity contained serous fluid—the left about a pint, the right less. There was also a moderate quantity of fluid in the peritoneum. Lungs: The right—upper lobe emphysematous; lower lobe nearly solid from large hæmorrhagic extravasation, not very recent, and undergoing inflammatory change. Several smaller and more recent spots of hæmorrhage were found in the same lobe. The left lung had the upper lobe emphysematous, and a recent large wedge-shaped extravasation in the lower lobe and its anterior angle. The liver was firm, granular, undergoing contraction; weight, 47 oz. Kidneys enlarged and congested. Spleen and other viscera healthy. The heart showed a well-marked white patch on the anterior surface near the apex; all the cavities were full of uniform black blood-clot. Weight of organ when emptied, 18 oz. Both auriculo-ventricular

orifices were dilated—the right admitting four fingers and the thumb, the left four fingers. The tricuspid, mitral, and pulmonary valves were healthy, but the papillary muscles of the mitral valve were thinnish and undergoing degenerative changes near their apices. The aortic valves, tested by water, were to a large extent incompetent. When further examined, this incompetency, as you can now see, was found to be due to a rupture of the right and left coronary segments at their angles of attachment. The right coronary segment was torn down five-sixteenths of an inch at its angle of attachment to the posterior segment; the other angle of attachment of this segment, together with the neighbouring angle of the left coronary segment, were torn down to almost the same depth (five-sixteenths of an inch). The angle of attachment of the left coronary aortic segment to the posterior aortic segment was also torn down about one-sixteenth of an inch. The right coronary segment, which was the most incompetent, had its anterior angle of attachment torn towards the corpus arantii, but contracted, thickened, loose, and retroverted towards the ventricle, so as to flap idly in the blood-current. The corresponding half of the left coronary segment was attached to the wall of the ventricle on a level with the attached part of the right coronary segment, and was thickened and

sloped obliquely from the middle of the valve to its attachment. The other half of this segment, although slightly torn down as described above, was healthy and competent. Both the ventricles were greatly dilated, and to a less degree their walls were hypertrophied, and undergoing degeneration. The aortic orifice measured three inches and one-eighth in circumference. The extreme length of the left ventricle was four inches and a half, while its greatest diameter was three inches and a quarter. The walls were three-eighths of an inch thick near the apex, eleven-sixteenths of an inch near the middle, and three-quarters of an inch at the base. The aorta showed patches of recent atheroma near the valves, but in other parts was healthy.

The phenomena observed during life, and the facts of the man's history, were thus found to be consistent with the conditions discovered after death. The valvular injury was clearly traceable to the accident, and occurred to healthy valves. The few weeks which had elapsed since his medical examination for admission to the American service, as well as his previous good health, strongly corroborate this view. I refer to it because some of you have seen with me lacerations of valves previously softened by degenerative change. Only last week, indeed, we had a specimen of such a rup-

ture. The laceration of a valve previously healthy is much rarer, and has special interest for us in the manner of its occurrence, in the symptoms which it occasions, and the effect it has on the duration of the patient's life. These points will demand our attention later on, but first I wish to make a remark or two on the murmurs we heard in the case of H. The systolic murmur heard at the left apex was due—as was held during life—to regurgitation through the enlarged auriculo-ventricular orifice, and was a necessary consequence of the dilatation of the ventricle, and the weakened and degenerating papillary muscles. The aortic murmurs are explained by the condition of the aortic valves. The systolic murmur was caused by the loose valve segment, which gave the special blowing and flapping character to the murmur. Twice previously in other cases of ruptured valves I have noticed similar characters under similar conditions; and this peculiarity may possibly guide you to a correct opinion in these cases. The diastolic murmur finds its explanation in the utter incompetency of the valves to act as stop-valves. There is only one feature of it on which I would dwell: it is this—the diastolic murmur was not propagated towards the heart's apex, but was propagated towards the ensiform cartilage, and was more audible over the right ventricle, than over the left

into which the reflux occurred. This, indeed, is the rule as regards the conduction of diastolic aortic murmurs. Only a few days ago, however, we had a case of aortic insufficiency in which the diastolic murmur was very distinct at the apex—equally, if not more so, than over the ensiform cartilage. How are we to account for this variation? In this last case the post-mortem, as I predicted, showed that the regurgitation took place through *incompetency of the posterior aortic segment*. The regurgitant blood-column fell on to the upper segment of the mitral valve, as we could prove by experiment after death, and as the thickening of the upper segment of the mitral valve showed it had during life. This, then, as I have several times shown to you in other cases, is the explanation of the conduction of a diastolic aortic murmur to the left apex. It depends on the regurgitation taking place through incompetency of the posterior aortic segment, either at its right angle or through perforation of its curtain. My attention was first called to this by the following instance of rupture of this segment through effort, which I published at the time in the *Pathological Transactions*, vol. xviii, p. 49.

M. W.,[2] aged thirty-three, a discharged soldier, was admitted into the Queen's Hospital, Birming-

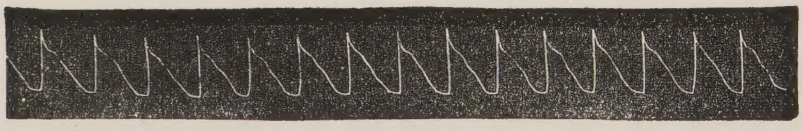
(2) Reported by Mr. (now Dr.) James Sawyer, clinical clerk.

ham, in September, 1866. He stated that he had always been in good health until the beginning of the last year, and he referred his present illness to an accident which had occurred to him at that time. For the last six years he had served in an Infantry regiment on the Mediterranean stations, and when quartered at Gibraltar in January, 1865, he injured himself in the following manner. One day when engaged in the cook-house, he was obliged to stretch across some high coppers to obtain a vessel, and in making an extraordinary effort to reach it, he was suddenly seized with an intense pain in his chest. As far as he can remember, he neither fainted nor vomited; but was carried to the hospital, where he remained for four days, suffering from pain in the præcordial region, palpitation, and dyspnœa. About ten months subsequently, the palpitation and difficulty of breathing, which had never completely left him, became much worse, and he was again obliged to enter the military hospital. After five weeks' treatment he was sent to England, and was discharged from the army in January, 1866; he has suffered ever since from difficulty of breathing, &c., and has been quite unable to work. Neither since his discharge, nor at any time previously, has he had rheumatic fever.

The following notes were taken a few days after his admission into the Queen's Hospital:—

His expression is anxious, there is slight duskiness of the lips, and the face is a little puffy. He complains of palpitation and dyspnœa, also of a fixed pain at the ensiform cartilage, whence a sharp pain shoots across his chest, and occasionally runs down his arms, particularly the left; this pain frequently occurs at night, and is accompanied by difficulty of breathing so great that he is obliged to sit up and struggle for breath. He has a harassing cough and expectorates mucus frequently mixed with blood. The urine is scanty and high coloured, but free from albumen. The legs are slightly œdematous.

Pulse 115, regular, jerking, and visible in the radials and larger arteries. The pulse trace as figured below (Fig. 1), shows the characters of moderate aortic regurgitation.



(Fig. 1.)

The chest expands freely, and is resonant all over the lungs. The breathing is rather harsh, and is accompanied at the bases of the lungs by small moist sounds.

The area of the cardiac dulness is somewhat increased, extending from the nipple to the right edge of the sternum. The apex beat is feeble

and close to the upper margin of the sixth rib, or one inch and three-quarters vertically below the nipple. At the base of the heart two murmurs are heard; that with the systole is somewhat harsh, and is followed by a long, soft, blowing murmur, which replaces the second sound and is terminated by the succeeding first sound. These murmurs are best heard at the junction of the fourth left costal cartilage with the sternum; they are also audible at the ensiform cartilage, and at the manubrium sterni. The systolic murmur is well heard along the aorta and in the carotid arteries, but it is not distinct at the back of the chest. At the apex of the heart a soft blowing murmur accompanies and follows the first sound. This murmur is well propagated towards the axilla, and is followed by a murmurish and prolonged second sound, which frequently amounts to a distinct murmur.

At first the man improved under treatment, but towards the beginning of November the dyspnœa increased; the œdema of the extremities became much greater, and blood again appeared in the sputa. The symptoms steadily increased in severity in spite of treatment; fluid was effused into the cavities of the chest and abdomen; and the patient died rather suddenly on November 24th. The *post-mortem* examination was made on November 27th.

A considerable quantity of clear serous fluid was found in the right pleural cavity, and some few ounces in the left. Both lungs were œdematous, and very highly congested, with spots of extravasation in the lower portions. The pericardium contained about twelve ounces of clear serous fluid. The heart weighed fourteen ounces and a-half avoirdupois. The right cavities contained some dark clots, the left but little blood. The aortic valves permitted free regurgitation. Both ventricles were dilated and hypertrophied; the cavity of the left was especially enlarged, but the thickening of its walls was not very great. On examination, the incompetency of the aortic valves was found to be due to the following conditions:—

The right angle of the posterior (or mitral) semi-lunar segment was torn from its attachment, so that the valve projected towards the ventricle; it was, however, still attached about one-quarter of an inch below the attachment of the posterior angle of the right coronary segment. The curtain of the injured valve was perforated in two places, each aperture being about the size of a small pea, while the central part of the curtain was pierced by smaller holes; as seen in the woodcut (Fig. 2). The other aortic segments were thickened, and rather closely applied to the aorta, in this respect contrasting with the projection of

the injured one. The thin edge of the left coronary segment was perforated by two small apertures, formed most probably by the absorption of the thinner part of its curtain. The superior part of the mitral valve was thickened at its free

(Fig. 2.)

edge on the auricular surface; the inferior curtain was healthy, as were the valves on the right side of the heart. The lining membrane of the aorta was thickened by atheroma; the aorta was small; and the circumferences of the aortic, pulmonic, and left auriculo-ventricular apertures, were less than normal. The liver and stomach,

especially the latter, were much congested; the spleen was small and unusually hard; the kidneys and other viscera were healthy.

The murmurs heard in the case just narrated find a ready explanation in the valvular conditions described. The murmurish second sound, however, was somewhat puzzling; at times it was a very distinct murmur, and could be clearly separated from the mitral systolic murmur. Though diastolic in time, it differed from a diastolic mitral murmur *in diminishing instead of increasing in intensity up to the first sound.* This character enabled me at the time to diagnose it as an aortic diastolic murmur conducted to the apex; and to disregard the hypothesis, that it was due to mitral stenosis. The examination after death showed that it was really produced by the insufficiency of the posterior aortic segment and perforation of its curtain, which allowed the regurgitant current to fall on to the mitral valve, and the murmur was thus carried with the stream so that it was heard in the same position as mitral murmurs usually are. This point, then, I wish you to remember, viz., that an aortic diastolic murmur, propagated to the heart apex, usually means incompetency of the posterior (or mitral) aortic segment. I believe we may also say that a similar murmur propagated towards the

ensiform cartilage, indicates incompetency of either the left coronary or the right coronary segment, by which the regurgitant current is thrown more upon the septum of the ventricles. I dwell upon this point because, as we shall see soon, it is not merely a matter of curious diagnosis, but has a possible and I think important bearing on the prognosis in this form of valvular defect.

I would now call your attention to the mode in which the valvular mischief occurred in the case of H. Clearly it was the result of the accident. The ruptures took place in valves till then sound, under the influence of the effort which the man made to save himself, when thrown forward on the deck. In all the cases of this kind on record it is some violent bodily effort—made generally with the chest well filled with air, and with its walls fixed—that gives rise to the injury. Under such circumstances, the tension in the aorta is at its highest, and the strain becomes too great for the delicate valvular folds. So it was in the case of M. W., who, stretching across a copper with his abdomen compressed, made a violent effort to seize one of his cooking-utensils placed on a shelf opposite; so also it was with S. (case below), who ruptured his aortic valves in endeavouring to throw an unusual weight of coal. In the case of H. we have two elements in the accident—the man's effort to save himself by grasping the side of the

hatchway, and the fall on to his chest. It is impossible to say to which of these elements the accident must be referred, but it seems to me, from a study of the cases on record, that the muscular effort was more probably the cause than the blow.

In most of the cases this accident has been followed by well marked symptoms, but from H. we obtained no clear history of how he suffered immediately after the rupture. There can be no doubt that such a rupture as he experienced must have produced very marked symptoms. We cannot conceive a considerable imperfection of such important valves occurring suddenly without some effect on the circulation. *A priori*, we should look for a temporary disturbance of the balance between the contents of the arterial and venous systems, with engorgement of the lungs and general venous congestion, which would pass away as the heart became more adjusted to the altered conditions of its work. The following case, which some of you may remember, gives us a very clear illustration of the immediate effects of such accidents:

Thomas S.,[3] aged thirty-three, stoker, was admitted into the General Hospital on August 22, 1870, apparently dying. He was cold, pulseless,

(3) Reported by Mr. H. C. Moore, clinical clerk.

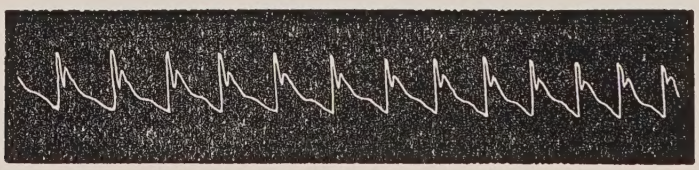
livid in the face, with skin covered with cold sweat. The House-Physician, Dr. J. B. Welch, diagnosed great congestion of the lungs, and very properly bled him from the arm. The blood flowed with difficulty, but he rallied a little, and he was placed in bed in warm blankets, hot bottles were applied to his feet, and sinapisms to his chest and legs. As soon as he could swallow, hot brandy-and-water was administered, and afterwards fifteen minims of ozonic ether every quarter of an hour. At the end of four hours he showed decided signs of reaction, and the alarming symptoms gradually subsided. He coughed a good deal, expectorated bloody mucus, and complained of difficulty of breathing and acute pain in the præcordial region. On the morning after his admission a double aortic murmur was detected.

On inquiry we found that his family history was good; that he had never had any serious illness, and no rheumatic fever. When about eighteen years old he enlisted, and served in the Crimea, through the Indian Mutiny, and in China where he suffered from rheumatic-like pains in the legs but was never laid up. He stated that he had never had syphilis and had always been fairly temperate. After leaving the army he became stoker at a gas-works, and when there he had a bronchitic attack, but was not confined to bed. He afterwards went to the axle-works at

Saltley, where he was at work when seized with his present illness. The mode of the attack was as follows: whilst lifting and throwing, in his capacity as stoker, an unusually great weight of coal, he felt a sudden and severe pain in the epigastrium, which stopped his breath and made him gasp for a moment or two. His head went light and everything looked dim, he fell down, and some of his fellow-workmen brought him some brandy, which revived him for a moment, but after this he remembered nothing until he began to recover in the hospital.

He mended day by day, but still complained of his breath and cough. About a week after the accident the following notes were made: Lung percussion, which had daily improved posteriorly, had become at this time healthy all over the chest. The breath-sounds were normal everywhere, but were rather more feeble over the right lung posteriorly than over the left. Heart: the apex-beat rather forcible below the sixth rib, just inside the nipple line. The cardiac dulness extended laterally from the nipple line to the right edge of the sternum, and from above the fourth rib to below the sixth. On auscultation there were heard at the base two murmurs, one systolic, the other diastolic. The systolic murmur was soft, short, blowing, and faintly flapping in its character, and was loudest at mid-sternum opposite the

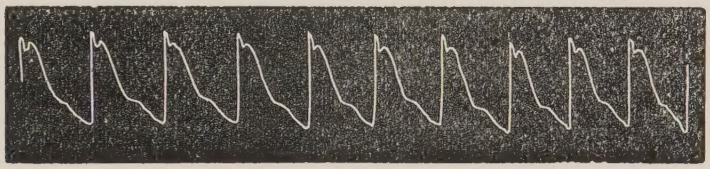
fourth rib; it was not conducted upwards very distinctly, and was inaudible at the apex. The diastolic murmur was loud, creaking almost in its character, masking second sound, and extended quite through the long pause. It was most distinct at the junction of the second right costal cartilage with the sternum, and opposite the fourth cartilage. It was loudly heard all down the right edge of the sternum, from the manubrium sterni to the ensiform cartilage. It was louder under the right clavicle than under the left, and could be heard in the carotids replacing the second sound, and was audible all down the spine posteriorly. It was not heard well at the apex; there it was faint and more indistinct than midway between the apex and the ensiform cartilage. At this time the radial pulses were jerky, and gave the following trace (Fig. 3) with the sphygmograph.



(Fig. 3.)

The pulsations of the carotids and the brachials were visible, though less so than three weeks later, when he requested his discharge. When discharged the cardiac dulness had decidedly increased, and extended as low as the seventh

interspace; there was also marked epigastric pulsation. The heart-sounds had not altered, except that a more purring than creaking character was noticed with the diastolic murmur, and a distinct thrill could be felt during the diastole over the sternum and the base of the heart. The impulse was heaving and diffused; the pulse more bounding and of higher tension, yielded the following trace (Fig. 4), which shows clear signs, when compared with the former trace (Fig. 3) of the increased power of the left ventricle.



(Fig. 4.)

The liver projected one inch and a half below the costal arch; the splenic dulness was normal; the urine free from albumen, but scanty and loaded. The expression of face was anxious, and the colour somewhat paler and sallower than natural. He had been kept on a generous diet during the last fortnight of his stay in the hospital, and had taken—in addition to occasional purgatives—a mixture containing per-chloride of iron and chloric ether. He was directed to live as well as he could, to take a fair amount of meat, and to continue his medicine.

Only two months later he was brought into the hospital moribund. The legs were greatly œdematous; the breathing was laboured; both lungs were congested and œdematous; the heart's action tumultuous, and the beats irregular and unequal; the pulse, with same characters, was thrilling, and of low tension and beat 104 times a minute; the face was dusky. He was, in short, in a state of asystoly, from which he never rallied, and died eighteen hours after admission. We learned from his wife that he had tried to work after his discharge, but had been unable to do any heavy work, and consequently maintained himself by odd jobs, and at times had been very short of proper food. He had got worse daily for two weeks before his re-admission.

The post-mortem examination disclosed the following points of interest: On opening the thorax, the right lung was found to be adherent. In the left pleura there were some seven ounces of fluid. Both lungs were congested, and in both were spots of recent hæmorrhage. The liver was large, and weighed four pounds six ounces. Both the kidneys were congested, and the spleen also, but not much enlarged. There were twenty ounces of fluid in the peritoneum. In the pericardium were three to four ounces of serous fluid. The heart was greatly dilated and enlarged, and weighed twenty-one ounces. All its cavities were

full of blood-clot, fibrinous and black. The right cavities were dilated, but their valves were perfect. The left cavities were also dilated, especially the left ventricle, which was greatly so, while its walls were not hypertrophied to a corresponding extent. The mitral valves were somewhat thickened, and were less transparent than in health. The mitral orifice was dilated, and admitted four fingers. The aortic valves were incompetent. The incompetency was caused by lesion of the right coronary segment, which was torn from its attachments, and had its free margin thickened and retroverted towards the ventricle. At its angle of attachment to the left coronary segment it was torn down five-sixteenths of an inch; and the chief regurgitation seemed on testing to take place through the chasm thus formed between these segments. The other attachment in connexion with the posterior or mitral segment was torn down to an equal extent; but the posterior segment overlapped the gap thus created, and partially prevented the regurgitation at this point. The posterior or mitral segment was torn slightly at its left angle of attachment to the extent of about one-twentieth of an inch, and the valve itself was softened, and in a state of degeneration. The aorta was atheromatous to a moderate degree above the valves—no doubt as a consequence of the injury, since higher up and throughout its

course it was healthy. The cardiac muscle (left ventricle) was undergoing fatty degeneration: this was especially the case with the papillary muscles.

This case teaches us very forcibly that the symptoms of a suddenly-created imperfection of the aortic valves are what we might *à priori* expect. Here, crowded into the space of a few seconds, we had all the phenomena of obstructed intra-cardiac circulation, which generally take months to develope. When the rupture occurs, the immediate effect is for the blood to pour through the rent into the left ventricle. That cavity, surprised, as it were, by the novel rush of blood from the aorta, in addition to the accustomed stream from the auricle, staggers under the load, falters in its action, and hence the syncope. Even when the ventricular muscle rallies again to its work, it cannot for a time cope with the increased blood-charge. Whatever flows back from the aorta must keep back some of the contents of the auricle, and so distend that cavity, and soon congest the lungs. The ventricle dilates, however, under the extra blood-pressure, and thus throws at each systole into the aorta a larger quantity than the normal charge. In time this extra charge becomes large enough to allow for the regurgitation, and still to keep up an approximation to the normal contents of the arterial system. For a time a healthy car-

diac muscle would be competent for this increased effort; but its reserve power would not last long, and its nutrition would suffer: therefore the next change soon occurs. Not only does the cavity dilate to contain more blood, but the walls thicken, so as to propel the larger quantity with sufficient power. This hypertrophy renders the left ventricle a more equal match for the valve defect, and when the hypertrophy and dilatation are so nicely adjusted to the lesion that each ventricular systole propels into the aorta a charge of blood sufficient to allow for the reflux, without robbing the arterial system of its due, the compensation is practically perfect, and the normal balance between the arterial and venous contents is maintained.

In this way the compensation was effected in both the cases before us. How comes it, then, that it was only temporary? To answer this question we must consider the mode in which the heart is nourished. Working as the cardiac muscle does constantly, with only that brief rest which it obtains between each systole, its nutrition must be most active, in order to support the wear and tear of its constant work. This active nutrition is provided for by the position of the coronary arteries, which, rising from the first part of the aorta, are so situated that for each cardiac contraction a double wave of blood enters them. They receive some blood from the

cardiac systole, but the aortic systole fills them more thoroughly, causing the blood to bound, as it were, from the tense aortic valves into the open mouths of the coronary arteries. It is no doubt this second or diastolic wave which gives the heart most of its blood-supply; for it is during this period of diastole, when the ventricular muscles are relaxed, that the nutrient current can circulate most readily through the ventricular walls. Now, in all forms of imperfect action of the aortic valves, the rebound of the blood-column from the closed valves is weakened, and therefore the diastolic blood-wave is sent into the coronary arteries with less force. In all these cases, too, the aortic tension is lessened by the regurgitation, and the less the tension in the aorta, the less perfect is the implétion of its coronary branches.

These are the reasons why all cases of aortic insufficiency are sooner or later followed by failure of the cardiac muscle. The work for the ventricle to do is excessive, the nourishment it receives is defective, and the inevitable consequences are—muscular degeneration and failure of compensation. Many cases of aortic insufficiency, however, go on for years; the compensation is maintained so perfectly that scarcely any inconvenience results. In all such cases that I have met with, the valve-lesion has been the consequence of disease,

and not of rupture from accident; and I think I may also say the regurgitation must have been slight. In the cases of insufficiency from disease the valves are often increased in size and substance by inflammatory products, and the insufficiency is due to a want of perfect apposition of the valves at their edges of junction. In cases of rupture of healthy valves, on the other hand, the lesion is generally greater, and by its position at the base or attachment of the valve allows more free regurgitation. This is, I believe, the reason why in these cases of ruptured aortic valves the patients live so short a time after the accident. In no instance, as far as I can discover, in which rupture has been found after death, has its occurrence been referred to a date more than four years and a half previously. In the three cases which I have brought before you, the duration of life after the accident was three months (S.), about eighteen months (H.), and about twenty-three months (M. W.). Between the longest period and the shortest there is a considerable difference, due, I have no doubt, mainly to the extent of the lesion, but partly to the conditions under which the patients were placed after its occurrence.

The seat of the rupture has, however, I am strongly inclined to believe, a considerable influence in determining the duration of life. Two of the segments have above them each a coro-

nary artery, which is filled by the blood-column as it rebounds from their curtains. When these segments are torn down and retroverted, the regurgitant blood-current running past the mouths of the coronary vessels must to some extent diminish the amount of diastolic blood-wave which they receive, and consequently impair the heart-nutrition. When the aortic segment immediately below the coronary artery is imperfect, the filling of the artery, probably both at the time of the ventricular systole as well as at the time of the aortic systole, must be impeded. It was to this point that I alluded when I said that if my explanation of the propagation of an aortic diastolic murmur to the left apex were correct, this was not a matter of mere curious diagnosis, but had a bearing on prognosis. That segment of the aortic valves, by whose incompetency, we believe, a murmur is specially carried to the left apex, has no coronary artery above it, and therefore, when it is affected, we should expect the coronary circulation to suffer less than when either of the other segments is imperfect. The coronary arteries, when this segment is torn, would have no regurgitant current running at right angles close to their orifices, and no thickened valve to divert the systolic wave from their mouths, and would suffer only in proportion to the general loss of tension in the aorta. As far, then, as the coronary cir-

culation is concerned, imperfection of the posterior or mitral or non-coronary aortic segment should, *cæteris paribus*, be less serious than a similar imperfection of either of the other segments. There is, in my experience, reason to believe that this is so. Of the three examples of rupture of these valves to which I have referred to-day, that patient, in whom the non-coronary segment was the injured one, lived the longest time. In the other two cases in which the segments below the coronary arteries were torn, and in cases recorded by other observers in which similar injuries existed, the duration of life after the accident was less.

The hypothesis I have advanced contains, I think, the elements of the explanation of the rapid progress of those cases in which the coronary segments of the valve are the seat of the lesion; and the conduction of an aortic diastolic murmur to the left apex, pointing as it does to the incompetency of the non-coronary aortic segment, becomes an important prognostic indication. These views are based on a careful study of these and many other similar cases, and I am convinced that a like examination of the finer features and the position of valvular lesions will, in many other instances, give us the clue to the long or short course which a case of cardiac mischief has run. The investigation of such details will at all events improve our

powers of observation, and so far make us better practitioners, even if it should not—as I think it will—lead to a more far-seeing prognosis and a more scientific treatment.

VI.

THE SYNTHESIS OF ACUTE RHEUMATISM.

The lactic acid theory of acute rheumatism has of late somewhat declined in popularity. The arguments of Prout, the experiments of Richardson, and the statistics of the alkaline treatment, have all failed to establish it firmly. Like many other theories resting on surer grounds, it has suffered from the scepticism of the day. The mint water treatment and other similar manifestations of nihilism have served, more or less, to strengthen the bias in favour of doubt, and to increase the number of sceptics. For some years past I was myself a disbeliever in the lactic acid theory, and it is to lay before the profession the facts which have recently recalled me to my allegiance that this paper is published. These facts, when added to the arguments which have been adduced by many previous writers, and to the experimental proofs which we already possess, will, in my opinion, strengthen the evidence which

points to lactic acid as the poison, or one of the poisons in rheumatic fever. For the development of acute rheumatism, there is no doubt required an antecedent condition of defective assimilation, which depends most probably on altered nerve influence, but the facts here recorded show, I think, that the immediate antecedent of acute rheumatic symptoms consists in some cases of an excessive formation of lactic acid, or some similar product of disordered nutrition.

In the *British Medical Journal* of February 25th, 1871, I read with much interest an account of Dr. Cantani's observations on the lactic acid treatment of diabetes. At that time I was engaged in completing an inquiry into the effects of different drugs on the sugar-excretion in diabetes. I determined to add one more drug to my list, and to complete my research by observing the effects of lactic acid.

A man (Wright) who had just come into the General Hospital under my care, suffering from diabetes, offered me the opportunity. His age was thirty-one, and he had been ill some four months before his admission. By trade he was an ironcaster, and up to this attack of illness he had been a healthy man, and had never suffered from rheumatism. He was married, and had several strong, healthy children. On a mixed diet, he passed

during the first week of his stay in hospital an average of 180 ounces of urine daily, containing 49 grains of sugar in the ounce. On a strictly animal diet, continued two weeks, the sugar fell to an average of 36 grains an ounce, and the urine passed to an average of 116 ounces daily. The skin was dry and branny. The sugar-excretion remained pretty stationary on strict diet, but lung-symptoms began to manifest themselves, and steadily increased.

On March 8th, I ordered the patient fifteen-minim doses of lactic acid dissolved in an ounce of water four times a day. The dose was doubled the next morning, and in the afternoon he complained of acute pains in his joints, and flying pains about the limbs. In the evening, as these pains had increased, the medicine was discontinued by order of the resident medical assistant, Mr. E. A. Elkington.

On March 10th, no lactic acid mixture was taken, and the pains gradually ceased.

On March 11th I saw the case; and, regarding the occurrence of the joint-pains as a mere coincidence, repeated the lactic acid in fifteen-minim doses three times a day. On the evening of the 12th he again felt pains in his joints; and on the morning of the 13th, "the small joints of the fingers of both hands, the wrists, and, in a less degree, the elbows," were noted by Mr.

Elkington, to have become "red, swollen, and painful." On my visit, I was much struck by the appearance of these joints, which were typical specimens of acute rheumatic arthritis. In the evening, both wrists, the small joints of the fingers, and the elbows were all red, hot, swollen, tender, and painful. The heart-sounds were clear. The temperature in the morning was 100° Fahr.; in the evening, 101°. He had moderate perspiration. Pulse 90, soft and full. The joints were wrapped in cotton-wool, and the lactic acid was discontinued.

On March 14th, in the morning, there was a decided improvement in all the joints; the swelling had much diminished, but heat and pain were still present. Temperature, 100°; pulse, 84. In the evening all the small joints of the fingers were much better. The wrists were still affected, and he complained of a good deal of pain in the knees, which had hitherto escaped. The heart-sounds were clear. Pulse 90. Temperature 100°8.

On March 15th the joints were better. The temperature in the morning was 98°6; in the evening, 99°4.

On March 16th, he said that his arms were quite well, and his legs nearly so. He had slept much better.

On March 17th, all pains in the joints were gone. Temperature 98°2. Pulse 72.

During the next twelve days no lactic acid was administered. The probable influence of the lactic acid in producing the rheumatic symptoms was clearly explained to the man, but as he had felt benefit from the acid mixture and had passed less urine during its use, he decided to run the risk of acute rheumatism. Accordingly, on March 29th, I prescribed seventy-five minims of lactic acid dissolved in twenty ounces of water. This was to be taken as a drink in the course of each twenty-four hours. During the next five days no rheumatic symptoms appeared. The pulse rose twelve beats on and after the third day; the temperature, which had been previously elevated by the lung-complications, showed no marked change, but on the fourth and fifth days remained steadily at 99°, instead of varying, as it had done for some time previously. On the morning of the sixth day (April 4th), he complained of having had a bad night from joint-pains, which had disturbed him very much, and which came on suddenly after midnight. On examination, the metacarpo-phalangeal and first phalangeal articulations of the first and second fingers of each hand were found to be red, swollen, hot, and painful; the slightest movement aggravated the pain, and he could not on this account pick up anything with his fingers. The pulse was 102. The temperature, which on

the previous evening had been 98°2, had risen to 99°4. The heart-sounds were clear. The acid mixture was stopped, and in the evening the pain in the knuckles was less, and the redness had diminished; they were, however, still stiff. No other joints were affected. Temperature 99°2.

April 5th.—His hands were much better, and, of his own accord, he resumed his lactic acid drink, and took about thirty minims of acid in the course of the forenoon. In the evening, the pains had returned in the knuckles, which were swollen, red, and tender. He discontinued the acid, had a fair night, and on the morning of the 6th found his hands free from pain. He again resumed the lactic acid, and took up to 4 p.m. the remainder of the bottleful, containing about forty-five minims of acid. In the evening, at 9 p.m., the pain and swelling had returned in his knuckles, and his left wrist was also affected. He now gave up the acid for two days, and the joint-symptoms gradually disappeared.

The acid drink was resumed on the 9th, and continued to the 13th, but he only took about thirty-five minims of acid a day. He experienced no inconvenience, except flying pains about his joints, till the night of April 13th, when he was disturbed by severe pain in the right wrist, which was found in the morning to be red, swollen, painful, and hot, and was a typical specimen of a rheu-

matic joint. Pulse 98, full and soft. There was copious perspiration, of acid reaction. The heart-sounds were clear. The elbows and knees became painful and stiff the next day. The joints were all wrapped in cotton-wool as before; and in the course of four days nothing remained except a little stiffness in the right wrist. After a week's interval the acid was again taken with like results.

The man now had gained so much experience as to the first indications of a coming attack in his joints, that he was allowed discretionary power as to the time and manner of taking the mixture. By trying it first in small doses, so as not to take more than twenty minims of acid a day, and stopping it for a day or so whenever the joints threatened to become painful, he managed to continue the acid for some weeks. Gradually he increased the dose, as advised, and early in June was able to take from forty to fifty minims daily. During this month, he had two sharp attacks of rheumatism in the hands and wrists. By the end of June he was taking seventy-five minims of acid daily, and on July 6th this was increased to 100 minims. On the 7th he began to experience considerable pain and stiffness in his joints, and kept his bed (he had previously been up daily) on account of the pain caused by walking. On the 8th these symptoms were worse, and in the evening his wrists and elbows were very stiff and painful, but

the knees were less so. The temperature had risen to 100°6. The acid was stopped. On the next morning he was better. Temperature 99°. The joints were less painful and stiff; there was no redness and no swelling. On the 10th he again took the acid, his joints feeling much better, the temperature being only 98°4. In the course of the day he took 100 minims of the acid; and by the evening the pains had returned in his wrists, elbows, and knees. Temperature 100°6; pulse 100, full and soft; skin moist and perspiring. On the morning of the 11th his right wrist was red and swollen; the left less so. The knuckles of his right hand were also red, swollen, and painful. His left knee was red, swollen, and very painful and tender. He complained also of pain in the left side, but the heart-sounds were found to be clear; pulse 88; skin still moist. The mixture, which had been stopped on the previous night, was discontinued till July 17th, by which date all the rheumatic symptoms had subsided. After this the man only remained in hospital seventeen days. During this period he, of his own desire, resumed the acid drink, and on one occasion took as much as 125 minims of acid in the course of twenty-four hours. During the last fortnight of his stay in hospital he had no severe pains in his joints, and whenever flying pains warned him he discontinued the medicine for a day.

While the above case was under my care in the hospital, it so happened that another diabetic patient of mine, in visiting the wards, met Wright and compared notes with him. From him he heard such a favourable report of the acid treatment, that he requested me to order him the same medicine if I thought it suitable. I did so. A drink consisting of seventy-five minims of lactic acid in a pint of water was prescribed. Of this he took daily as much as contained thirty to fifty minims of acid; and on the fourth day he came to me complaining of a sharp pain in his right knee, which rendered the joint stiff, and made walking very painful. He also mentioned that he had less severe pains in his other joints, and expressed his opinion that he had caught a cold, which had produced rheumatism—a disease from which he had never before suffered. There was no swelling or redness of the knee or other joints. His skin, which had hitherto been harsh and dry, was soft and moist. The acid mixture was discontinued, and in two days the pain had entirely ceased. During the next month he made several attempts to take the acid mixture, but it was always followed in a day or two by pains in the joints. Early in May he managed to take the mixture for a week, and then was laid up with such severe joint-pains that I was called to visit him, and found him in bed with pains in his elbows,

shoulders, ankles, knees, and, as he said, all over him. None of the joints were swollen except the right knee, which was faintly red, decidedly swollen, and very tender and painful. The other joints were simply stiff and painful on movement. The skin was freely perspiring. Pulse 96, full and soft. The acid mixture was stopped, the joints were wrapped in cotton-wool, and alkalies administered. In the course of a week, all the symptoms had disappeared, and the patient was able to walk about and resume his ordinary habits. This patient had never passed more than twenty-four grains of sugar an ounce while under observation. The excretion was generally not over fifteen grains an ounce.

The above record contains an account of the joint-symptoms which were observed in two cases to follow the administration of lactic acid. In the first case, at least six well-marked arthritic attacks occurred; in the second case, under conditions less favourable for observation, as to duration of treatment and place, one well-marked attack occurred. The phenomena corresponded in all respects to those which are characteristic of acute articular rheumatism. They came on when the acid was taken, and ceased when it was discontinued. When moderate quantities of the acid were tolerated, an increase in the dose was

succeeded by the painful inflammation of the joints. Coinciding with the development of the articular affection was the appearance of perspiration, at first only slight, but afterwards, in the more severe attacks, copious and acid.

These facts have dispelled the last lingering doubt in my mind as to the truth of the lactic acid theory of rheumatism. At first I questioned the connexion between the administration of the acid and the production of the rheumatic phenomena. In my scepticism, I regarded it as an accidental combination. The recurrence of the joint-symptoms, however, on March 13th, following distinctly on the repetition of the lactic acid mixture, shook my disbelief. The coincidence of joint-attacks with the use of the drug might occur once, and I thought even a second time; but, when I found it occur over and over again, there was no room left for the hypothesis of coincidence. To refer Wright's attacks to a series of accidental combinations requires, in my opinion, a much livelier faith than to accept the hypothesis that the introduction of an excess of lactic acid into the system is a possible if not constant factor in the production of acute rheumatism. If to some, Wright's case presents not evidence enough, in the beautifully typical character of the artificially produced disease, and in the precision with which it could be manufactured

at the will of the experimenter, then the second case comes in to refute any explanation founded on the assumption of an idiosyncrasy on the part of one patient.

In health lactic acid appears to be developed in considerable quantities by the action of a ferment on the glycogen and glucose formed by the liver and stored up in the muscles. Muscular action also produces this acid, and the decomposition of the muscle sugar accounts for the acid reaction observed in the muscles after death. Lactic acid afterwards by its combustion and conversion into carbonic acid and water gives rise to part of the heat of the body. The very high temperature [1] observed in some cases of rheumatic fever may very possibly be due to the combustion of the excessive amount of lactic acid in the system. Two of the most constant effects produced by the administration of lactic acid to diabetic patients are, in my experience, the elevation of the temperature of the body, and the restoration of the action of the skin. From the experiments of

(1) In the case of a gentleman whom I recently saw with Dr. Turton, of Wolverhampton, the temperature in the axilla rose during the last two hours of life from 109° F. to 111° F. These observations were carefully made by Dr. Turton, and the latter temperature, which was recorded a few minutes before death, was again indicated by the thermometer ten minutes after that event. The patient was a strong, muscular man, and had suffered previously from acute rheumatism; this last attack was characterised from the first by intensely acid and profuse perspiration, a highly acid state of all the secretions, and a very elevated temperature.

Schultzen [2] we learn that when the processes of oxidation are arrested in the organism a variety of lactic acid appears in excess in the urine. The accumulation of the acid in acute rheumatism may possibly be accounted for in this way, or as the liver troubles which so often precede the occurrence of rheumatism would seem to indicate, the liver-sugar may be faultily formed and consequently undergo conversion into the acid too freely or too rapidly, or again there may be too great activity on the part of the ferment which normally converts the sugar. By some one of these modes, or by a combination of them, the occurrence of lactic acid in excess may be explained, and possibly a more rational plan of dealing with the morbid process than we at present possess, may be discovered.

No doubt much larger quantities of lactic acid than any given in my cases would be excreted in health, without producing any perceptible disturbance in the bodily functions. The acid would escape by the skin, the kidneys, or, after oxidation, as carbonic acid and water. It cannot be justly argued that the quantities of acid taken by my patients were too small not to have escaped in this way. The conditions under which the drug was given must be borne in mind. In diabetes we

(2) Quoted in "Dr. Brunton's Lectures on Diabetes" (*British Medical Journal*, February 21st, 1874), which contain an excellent summary of our knowledge of the formation of lactic acid from glucose.

have a state of disordered nutrition very unfavourable to the conversion by oxidation of new compounds; and in Wright's case this was aggravated by the serious pulmonary complications. Associated with these, there was a dry and branny state of the skin highly unfavourable to the elimination of the lactic acid by one of the common channels. Lastly, the well-known persistent acidity of the urine in diabetes points to a pre-existing hyperacidity of the fluids.

These considerations are, I think, important, as defining the conditions under which the experiments were made—conditions most favourable to the development of the specific effects of the lactic acid. It was the combination of all these which rendered Wright so susceptible to the action of the drug. By the absence of one of them (the lung-complication), and the minor degree of glycosuria, we may probably explain the slighter susceptibility in the second case. The larger[3] doses of acid which Wright was able to take occasionally, towards the close of his stay in the hospital, find an explanation partly in his more careful management of the remedy, partly in an acquired toleration of it, and partly in the great improve-

(3) Since the above was written, I have given lactic acid in much larger doses: to the extent of more than two ounces daily, without producing any rheumatic symptoms. The effects of the drug were profuse perspiration and finally diarrhœa, which compelled its discontinuance. *Vide* Case XI, Article VIII.

ment which occurred under treatment in the state of the respiratory organs and in the sugar-excretion.

I refrain for the present from discussing the bearings of my observations on the therapeutics of rheumatism. The effects of the lactic acid on the excretion of sugar will be considered, with other modes of treatment, in a subsequent paper (Article VIII). In this communication, my object has been to lay before the profession facts which have an important bearing on the origin of a common and serious malady. If, by pointing out the nature of the poison of acute rheumatism, they help in the smallest degree to improve therapeutics, they will not have been observed in vain.

VII.

DUCHENNE'S PARALYSIS.

(PSEUDO-HYPERTROPHIC PARALYSIS.)

GENTLEMEN,

You have lately had the opportunity of observing three cases of a peculiar form of paralysis, which you have often heard me speak of in the wards as Duchenne's paralysis. Dr. Duchenne (de Boulogne), to whom we owe our chief knowledge of the disease, has called it *Paralysie musculaire pseudo-hypertrophique*, or *Paralysie myo-sclérosique*,[1] and the English equivalent of the first name is probably the best descriptive title of the affection; though I prefer to call it after the name of the distinguished observer who has described it so well. The history of these cases—very typical ones—I will now read, and we shall then be in a position to recognise clearly the special features of the disease, and to learn in what respects it differs

(1) *Archives Générales de Médecine.* Vol. I. 1868.

from other conditions with which it has been hitherto confounded.

CASE I.—Some three months ago a man brought a dull, strumous-looking boy (W. M.), aged nine years, into the out-patient room, to be treated for a weakness of the lower limbs. He stated that the boy walked imperfectly; that he often fell down, and could not raise himself again without assistance, and could not get upstairs; "but," said the father, "his health is good, and his limbs are not wasted—he has capital legs." These statements attracted my attention. The boy was stripped, and then the gastrocnemii and glutei muscles were seen to be greatly enlarged. They were firm to the touch, and stood out in bold relief when contracting. As the boy stood erect, the lumbo-sacral curve was exaggerated, and the legs were widely separated. His gait was waddling and straddling, the body balanced on each leg alternately; and as he walked gingerly on his toes, the heels never touching the ground, the effect was most grotesque. The boy was admitted, and the following history obtained from his parents, who are both healthy, have never had syphilis, and have no history of paralysis in their families. The patient is one of eight children, of whom one died in infancy of scarlatina. Two boys older than the patient are strong and healthy, and a boy and two girls younger are also

healthy, but the youngest is weakly. The mother says the boy in the hospital was a healthy, well-developed baby; that he has never had convulsions or any kind of fits; and has never been ill except with diarrhœa (which he had badly) and measles. He was, however, very weak on his legs, as an infant, was quite two years old before he walked alone, and for a long time afterwards was always stumbling and falling as he walked. He improved in this respect, and was able when six years of age to walk nearly a quarter of a mile, resting frequently and supporting himself by the wall. He was sent to school, but was very dull; and finally was allowed to remain at home, as the other boys frequently pushed him down in order to observe his grotesque and futile efforts to raise himself. He was never able to run or mount the stairs. When six years old his legs began to increase in size. His arms were always weak; he could never cut up his food like other children. His appetite was always good. He never lost power over the bladder or the rectum.

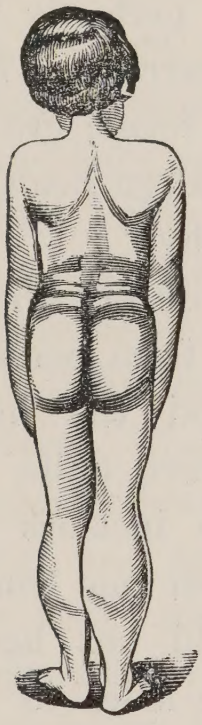
In the hospital the boy was found to be dull in intellect, but cunning; his speech was slow, his hearing slightly defective, and his vision myopic. The physical examination of his chest and abdomen detected nothing abnormal. The urine was healthy; the bowels regular. The appetite was good; during his stay in the hospital he gained

flesh. The teeth were irregular; the central incisors slightly serrated at their margins; the decayed stumps of the first lateral incisors unshed. On examining the locomotor system, the integument of the lower extremities was noticed to be remarkably mottled, patches of purplish colour alternating with white. This condition did not extend to the trunk, and was but slightly marked in the upper extremities. Sensation was everywhere natural; and he readily discriminated heat from cold. The bones and joints were well formed. The muscles of the head and neck were apparently healthy; those of the upper extremity were small and flabby, especially the flexor muscles of the arm and fore-arm. The pectorals were also small. The erector muscles of the spine were hypertrophied and hard. The glutei muscles were also greatly hypertrophied; the muscles of the thigh were firm, but not increased in size. The muscles of the calf were greatly enlarged; the body and the two heads of the gastrocnemii during contraction showed in bold relief beneath the thin skin which covered them. The calves measured eleven, the thighs twelve and a half, inches in circumference. The muscles of the calf contracted with considerable power, as estimated by the resistance they offered to flexion of the foot; while, on the contrary, the flexor muscles of the foot were easily

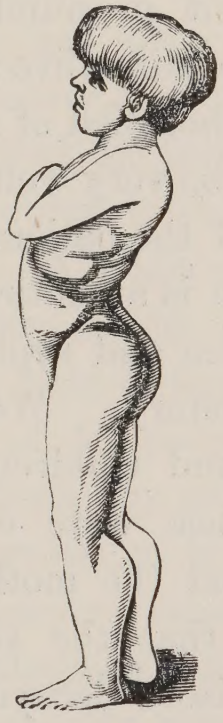
overcome. The muscles of the upper extremities were found to be feeble, the grasp weak on both sides, and the power of the bicipites small. When the boy stood erect the feet were separated ten inches or more; the heels did not touch the ground; the boy could not stand on his heels; even when supported, he naturally stood and walked on his toes. His equilibrium was so unstable that a very slight touch sufficed to upset him, and when down he could only occasionally raise himself without assistance. He generally crawled to the nearest bed in the ward, and placing his feet against the leg of the bedstead or part of the wall, as a *point d'appui*, raised himself first on his hands and feet, then, by placing a hand on each leg alternately, he, as it were, climbed up himself, and gained the erect posture, the spinal erector muscles bulging largely all the time, but being powerless to aid.

When he fell, he fell all in a heap; in sitting down he sat down suddenly, and could only rise again by using his arms. After walking across the ward two or three times the peculiarities of his gait were exaggerated, the efforts more laboured, and the perils of the journey increased. The photographs before you give a very fair idea of the boy's figure. (Figs. 1 and 2.) In one respect they are very imperfect: the feet are placed too close together. The artist when he brought the photographs to me, remarked that "it was a very

troublesome job, for the boy repeatedly fell down." In reality the artist had been trying to make the boy stand with his heels too close together, and after repeated trials had found it impossible to obtain any nearer approximation than that seen in the figures; whenever the basis of support was still further narrowed, the boy swayed about and fell.[2]



(Fig. 1.)



(Fig. 2.)

Such is a pretty full account of this case. The two other cases to which I have referred were under the care of my colleague, Dr. Russell, who has kindly placed them at my disposal, and allowed me to make use of his exhaustive and

(2) For the subsequent history of this case see Postscript.

graphic account of them. The second case I visited and examined with Dr. Russell at Cradley.

CASE II. "Zachariah W., aged eleven, was admitted April 5th, 1869. I visited his family twice in company with my clerk, Mr. Greenway. We saw the paternal grandparents, who have always been healthy, save that the grandmother has lately had an attack of hemiplegia. Their children, eight in number, were perfectly healthy; seven are still alive; no miscarriage. Maternal grandmother died of rheumatic fever; grandfather stated to be very well; five children healthy. The parents of the patient are healthy."

"There is an entire absence of any evidence of rheumatism and syphilis, both from personal and family history. We examined the hearts of parents and children; they were quite healthy. The corneæ were clear; the incisors healthy, except that the mother has lost one front upper incisor; that the second child has her upper front incisors decaying at the sides in common with other teeth, and the sixth child has her front incisors (first dentition) decaying. The parents have had nine children—one miscarriage. The eldest and fifth children died of inflammation of the lungs and of measles; the second child is now sixteen years of age; all are quite healthy and well-developed, except Thomas (the third) and our patient (the fourth),

both of whom are paralysed. No fits in any member of the family."

"Thomas, the third child, aged fourteen, was sickly as a child, but improved up to his eleventh year. About a year and nine months ago he sat down for two hours in a field by a bonfire; he was unable to get up, so that his mother had to carry him home. He has never walked on his feet above a yard from the door since. It is, however, evident that there had been weakness in walking anteriorly; moreover, he frequently wetted and once or twice dirtied his bed—a thing new in him. For the last twelve months he has only got along by walking on his hands and knees, for he could never use crutches, but he has ceased to wet or dirty his bed; his bowels have become very costive. The evidence of his parents is absolute against there having been any enlargement of muscles, and against progression on the toes. The boy is well made, his face very impassive, his pupils are rather large and sluggish, his intellect clear, but below par. He usually sits with his right leg bent under him, and manifests considerable weakness in the muscles of the back; as he lies his legs are closely bent on his thighs. In progression he goes on his hands and knees, advancing slowly, but with a perfectly symmetrical movement, just like a quadruped, his palms being placed flat on the ground, in the manner of a fore-

paw, indicating long practice in this method of locomotion. He is feeble in his upper extremities, but they suffice for ordinary purposes; he can place a book on his head. The trunk muscles, with those of the hips and back, preserve their normal proportion, but in raising himself after having stooped over the bed-side, want of power, both in his loins and in his arms, is plainly indicated. The muscles of the shoulders and upper extremities are extremely attenuated, so as to fix the attention at once by the disproportionate smallness of the arms. The lower extremities are of about normal bulk, and retain considerable contractile power. The muscles of the thighs are normal, but those of the calves are preternaturally firm, approaching, though not equalling, in this respect the calves of his brother. I shall give a description of the microscopic appearances presented by the muscular tissue of the calves, in connexion with the examination of his brother. The hamstrings are contracted, so that the knees cannot be fully extended, owing doubtless to his habitual posture. The integuments of the lower extremities are somewhat mottled. Temperature of lower extremities, 98.4° (normal) in six out of nine observations, in the other three from 96° to 97°. An interrupted galvanic current of moderate power produces contraction in the muscles of the lower extremities to about the same extent as in

the other muscles of the body, which are quite sensitive to its influence. Height of the boy 43 inches (not allowing for the flexion of the knees); girth of calves, 9 inches; thighs, $12\frac{1}{2}$ inches; of upper-arm and fore-arm, 6 inches. The muscles of the eyes, face, tongue, and throat act naturally. Sensation tested by a hair is natural. He readily discriminates heat from cold, but is not sensitive to tickling of the soles."

"Zachariah, aged eleven, our original patient, is a well-made boy. His corneæ are clear; the middle upper incisors have a transverse furrow; surface of lower half rather uneven; but no disposition to pegging. The heart is healthy. His mother is sure that he walked perfectly up to the last six months. At that time weakness in walking first showed itself, and has increased gradually, but up to a week before admission he could walk across the street, though when he came into the Hospital he was unable to walk a step without support, but he has now much improved in this particular. To the last he has been very useful with his arms, nursing the baby, &c. His mother declares that the boy walked on his toes from the beginning of the paralysis, and that the calves of the legs began at once to increase, and that there was also marked projection of the hips from the time when the failure was first noticed. "I thought his bottom was not right, it seemed so hard," she said. She

describes him very graphically as walking with his hand behind him, and his body swaying. He complained throughout that his legs ached, and said that the feeling had gone out of his hands, and that his feet were numbed. His bowels have not been costive, nor has he wetted or dirtied his bed. The boy has a sharp expression of face, and a state of intellect above the average. The pupils are quite normal in size and contractility. The muscles of the upper extremities and of the upper part of the body are of average size and proportion; but the nurse reports him as being rather clumsy in using his arms. The erector muscles of the spine in the lower half of the trunk, the muscles of the hips, and those of the calves, are remarkably enlarged and much firmer than natural. The hips project considerably; the erector spinæ is very prominent even as he sits, and the rounded calves—a roundness in which the muscles on the front of the leg participate—form very remarkable objects. The boy cannot raise himself from the sitting posture without help, nor straighten himself after stooping unless by climbing up his own thighs. In sitting little would be observed about him, but in standing or walking the pose of the body and limbs is very singular. The loins are excessively hollowed; the chest and abdomen are projected forward, calling to one's mind the look of a pouter pigeon; the arms are placed against the sides, and the

shoulders are raised; the legs are separated widely, and he supports himself on his toes, especially those of the left foot. He cannot stand more than a few seconds without support, and his entire weight soon falls upon the person supporting him. He can, however, now walk up and down the yard without help. In doing so the eyes are directed intently upwards; the muscles of the face are fixed; his lower extremities are straddled and straight; the knees are scarcely bent, the feet are extended; he progresses entirely on the balls of his toes, swaying his body with each step from one side to the other, and balancing himself with his outstretched arms. The peculiar waddle, the intent gaze, the fixed attention of every muscle of the face and trunk, the high shoulders, the projecting chest and belly, the pointed toes, form altogether a most singular picture. The integuments of the lower extremities are remarkably mottled, the skin is rather fine, the nails perfect. Temperature of lower extremities, 98.4° (normal), in five out of nine observations; in the remaining four from 96° to 97°. The muscles of all parts of his body alike did not answer to the induced current, contrasting remarkably in this respect with the results of two experiments on the brother; the current, however, was felt very sensibly. Sensation to a hair, to warmth, and tickling, is quite normal in

all parts. The muscles of the head, face, and throat act naturally."

"An ophthalmoscopic examination made by Dr. Welch, the house physician, gave these results. Zachariah: the fundus of each eye, on examination both of the direct and inverted image, appears normal, with the exception of a narrow crescentic white line running along the outer side of the disc. Thomas: fundus quite healthy."

Case III. "Samuel R., aged ten. The following is the remarkable history of this patient's family:— There is entire freedom from any rheumatic history. The hearts of the parents and children are healthy. There is no evidence of syphilis; the corneæ and the front teeth of all the children are quite healthy. The father's health and his family history are perfectly satisfactory. The mother is quite healthy. Her mother's brother (patient's great uncle) was "weak" (paralysed). She had heard her mother say that she had to carry him up to bed, just as she herself did for her own children. There were nine children of the generation of the patient's mother — one of them born dead: one miscarriage. Two of these children (patient's uncles) walked up to the age of nine years, and then became paralysed. They died at the ages of sixteen and seventeen. The rest were healthy. Three of them died young. The patient's mother has had nine children, one of them still-born. All were

stout and healthy at birth; none died under the age of five. The first and fourth children (boys) died paralysed, as will be related. The second and third died from inflammation of the bowels and fever. The fifth is our patient. All these were males. The remaining children (girls), eldest aged eight, youngest four years, are quite healthy. There have been no fits in any member of the family. The two boys who died paralysed began to lose power at the respective ages of four and nine. They never walked at all for six and four years before death. Both of them lost the use of their upper extremities also to such an extent, that in eating they had to rest their elbows on the table, and, finally, had to be fed. The one died, aged sixteen, of an acute attack in the bowels; the other, aged thirteen, of consumption (fever, expectoration, and wasting). Neither had large muscles; on the contrary, their limbs wasted, but the former of the two is positively asserted to have walked on his toes, and to have swayed his body."

"Samuel, the fifth child, aged ten, has clear corneæ, excellent front teeth, and a healthy heart. His legs first failed at the age of three or four, and he grew worse by degrees. They began to grow large at a very early period in the history of his disease, but have only become "so very large" the last four years. He has never fully lost the power of walking; has never had pains

nor any spasmodic action. He can now walk three or four miles, but is very easily thrown down, and when we saw him he had a bloody nose from such an accident. His walk, and especially his run, are unsteady. He stands steadily and well, but the heel is not fully placed on the ground. He has marked difficulty in stooping, and especially in raising himself again. The right grasp is decidedly more feeble than the left, and he is stated to fail somewhat in his arms, though he throws stones and spins tops. The muscular enlargement is confined to his calves, which also present unnatural firmness. In girth they are within an inch of the thickest part of the thigh, which itself is not reduced in size. In the thighs, however, the belly of the quadriceps extensor projects so much as to cause a very apparent deformity, but it is not firmer than normal. The arms are small, but the muscles are fairly nourished. Sensation to touch is perfect. Intelligence is stated to be good, but he is very shy and unmanageable before strangers."[3]

The diagnosis in all these cases was clear. The enlarged and hardened muscles, the history of previous weakness of the legs coming on without appreciable cause, the separation of the legs in the erect posture, the talipes equinus, the curved

(3) *Medical Times and Gazette*, May 29th, 1869.

back, and the peculiar gait, formed an assemblage of symptoms too complete to admit of a doubt.

Duchenne, in his admirable memoir on this subject, has divided the course of the disease into three stages :—

I. Of muscular weakness.
II. Of apparent muscular hypertrophy.
III. Of general paralysis.

I. The first stage is characterised by feebleness of the muscles of the lower limbs. The children walk late and walk badly; when they stand their legs are widely separated, the natural lumbar curve of the back is greatly increased; they waddle in their gait, and with difficulty maintain the erect posture. This stage varies in its duration; it is usually shorter than the subsequent stages: it lasted in our first case from the earliest infancy up to the sixth year. In the other cases it began later; in the one it lasted between two and three years, in the other scarcely as many months.

II. In the second stage the enlargement of the muscles makes its appearance. The muscles of the calf are the first to increase in size, then other muscles follow, especially the glutei and the lumbo-sacral muscles. The hypertrophy may indeed extend to nearly all the muscles of the body, and give the child the appearance of a little

Hercules. The enlarged muscles are firm and hard to the touch, and stand out prominently beneath the skin during contraction. It would seem that a clear separation of the first and second stages may be wanting, and the weakness and enlargement of the muscles may, by a curious association, appear simultaneously. In some cases the hypertrophy is inconsiderable, and may escape the attention of parents—as in the case of two brothers of one of the patients here. This stage may last for one or two years; the muscles enlarge for a time and then remain stationary, their power not being appreciably diminished by the enlargement.

III. In the third stage the muscular weakness is greatly aggravated. The patients can no longer stand or even sit upright without support. They have no power to alter any position in which they are placed. The upper extremities become so weak, and the power of raising the arm progressively diminishes to such an extent, that the food can scarcely be raised to the mouth. The hands, however, retain strength enough to play with small toys. The enlarged muscles sometimes melt away, but often to a late period retain both their bulk and strength. This is especially seen in the muscles of the calf. Those of the face and neck remain intact longest, and the well-nourished face and head contrast most curiously

with the useless limbs and the inert trunk. In this state these unfortunate subjects may live for a considerable time, till some intercurrent malady ends the scene. The boy whose case I have placed first is now entering this stage; his muscular power is decreasing, and since he has left the hospital, this diminution has been very decided (*vide* Postscript).

The question occurs—how is it that muscles, apparently so much increased in size, can effect so little? The muscles of the gluteal region (Case I, Figs. 1 and 2) were enormous; and yet the alternate balancing of the body on either side indicated feebleness in the middle and small gluteals. By means of the lumbo-sacral curve, the shoulders and upper part of the trunk were, in the erect posture, placed in a position posterior to the sacrum, and the line of gravity thus thrown backwards. When this curve was diminished, the boy fell forwards, and could only raise himself by resting his hands upon his thighs, and jerking himself backwards, the hypertrophied spinal muscles bulging all the while in impotent contraction. To those who first observed the disease, the dead-house seemed to be the only place where a satisfactory answer could be obtained; but some observers,[4] with an enterprising

(4) Griesinger und Billroth: *Archiv für Heilkunde*, 1865.
M. A. Wernich: *Deutsches Archiv f. Klin. Med.* t. ii, 1864.
M. H. Heller: „ „ „ „ t. i, 1865.

boldness, cut out pieces of muscle from some of their patients. Duchenne, however, adopted a more humane plan, and devised a little instrument —the "emporte-pièce histologique,"—by means of which specimens of muscle can be easily and almost painlessly obtained from the living subject. I procured this instrument; and my colleague, Mr. Bartleet, removed a specimen for me from the gluteal region. The piece of muscle was very pale; and after it was treated by chromic acid, as Duchenne recommends, the microscope showed us a considerable increase in the interstitial connective tissue, and a remarkable development of wavy fibrous tissue. The muscular fibre itself did not appear to be much altered: fat-globules were seen near to, but not connected with it. A second specimen was examined, and showed the muscular fibre almost wholly deprived of transverse striation, but with distinct longitudinal striæ, and surrounded by a quantity of wavy fibrous tissue,[5] very like yellow elastic tissue; a few fat-globules and small cells were also visible between the fibres. Heller observed this fat in greater quantity, and proposed on this account to name the disease *Lipomatosis luxurians musculorum progressiva*. The fat is most probably the characteristic feature of the last stage of the disease, as

(5) These appearances were figured in my original paper in the *Lancet* (May 8, 1869).

the increased amount of connective tissue is of the second. The examination of the muscles after death in the two recorded necropsies discovered similar conditions to those which examination of the living muscle disclosed.

In Dr. Russell's cases the results of microscopic examination were as follows:

"In the case of Zachariah, the boy with enlarged muscles, specimens were taken from the calf and the hip; they consisted of muscular fascicles and of foreign element in different proportions. The latter consisted of a membranous looking material without structure, of strands of different diameters, from $\frac{1}{1000}$ inch to double that size, exceedingly twisted and contorted, some without, some with a fibrous structure, and of fine curled single threads. The muscular fascicles were much more abundant in the calves than in the hip. In one specimen from the former situation the fascicles were in large numbers, even constituting the larger proportion; in another specimen, on the other hand, taken from the calf, we only saw two fascicles, one of them partially atrophied. In only one specimen (from the hip) were the transverse markings of the fascicles naturally distinct; in all the others they were either absent, or only visible here and there. The longitudinal markings were very delicate. In no fascicles was there any sign of granular or fatty

degeneration. I looked with great interest at the specimens from the rather firm calf of the boy Thomas, who had not had enlargement of the muscles. They presented precisely the same appearance with the specimens described above. The foreign element was generally even more abundant. In one specimen only were the co-existing muscular fascicles healthy in appearance; in the other, the transverse striæ were faint, and the longitudinal very fine."

Duchenne has figured (*Op. cit.*) similar appearances to those above described, and thus enumerates the changes which occur in the muscles—(1) loss of colour; (2) extreme delicacy, and in some few cases loss of the transverse striation of the muscular fibres; (3) diminution in size transversely of the muscular fibres; (4) decrease in the number of muscular fibres in proportion to the connective tissue hyperplasia. The appearance of interstitial fibroid tissue, Duchenne says, is not to be referred, as Eulenberg and Cohnheim thought, to empty sheaths of sarcolemma. In the first stage of the malady the transverse striation is often extremely delicate, and in this stage there may be a condition of muscle which gives rise to slight enlargement, but the true connective tissue hyperplasia does not appear till the second stage. The passage from connective tissue to fat occurs later on. The existence of numerous hypertro-

phied capillary vessels in the muscles was noted in the examination of the muscles after death in Bergeron's case.[6] The essential change, indeed, appears to be the increase of connective tissue. This we found equally marked in the brother of the boy who was the subject of Case II, who had never had decided enlargement of his muscles. The morbid process is always the same: a formative irritation, which produces first hyperplasia of the interstitial connective tissue, and later, fatty change.

The hypertrophy is possibly only an accidental condition of certain muscles, and not a necessary part of the pathological process. It is certain from Duchenne's observations, and from our own, that the hypertrophy bears no direct relation to the paralysis, but on the contrary, the enlarged muscles retain their power to an advanced period of the malady, when nearly all the other muscles are wasted and useless. It appears to me that the conditions which favour the connective tissue growth may also contribute to the nutritive activity in the muscular fibre itself, and so conserve the necessary strength in certain muscles, on which much work is thrown. In this sense the hypertrophy may even be regarded as conservative in its nature. I find

(6) Duchenne. *De L'Electrisation Localisée.* Troisième Edition, 1872, p. 615.

that Dr. Davidson[7] has expressed a similar idea, and considers the hypertrophy to be compensatory. He explains the localization of the hypertrophy in the calves and posterior muscles, by the fact that these muscles have the greatest amount of work to do in keeping the body erect and in walking. In the third stage of the malady in Case I (*vide* postscript), the posterior muscles of the leg retained a considerable amount of strength till a late period, and showed a much greater amount of electro-muscular contractility than the muscles which had never enlarged. The deltoid muscle of the right side, moreover, which had been used for raising the arm long after the left arm had become useless, was moderately hypertrophied, while the left muscle had not at any time undergone such a change. This hypothesis of the conservative or compensatory nature of the hypertrophy, is worthy of further investigation.

The above described microscopical appearances distinguish the affection from one with which it has been hitherto confounded—progressive muscular atrophy of childhood—in which the muscular degeneration is decidedly fatty. Progressive muscular atrophy, which is rare in childhood, begins by attacking the muscles of the face, and follows

(7) *On Pseudo-hypertrophic Muscular Paralysis.—Glasgow Medical Journal*, May, 1872.

a slow and descending course, dissecting out separate groups of muscles. The wasting is partial and irregular, and is in direct proportion to the loss of power. Infantile paralysis may also be mistaken for the disease under notice; but the sudden invasion, the occurrence of febrile symptoms, and the completeness of the paralysis, at first, are generally sufficient to distinguish it. The electro-muscular contractility is also impaired, while in the earlier stages of paralysis with apparent hypertrophy it is unaffected. Our observations coincide on this point with those made elsewhere. The peculiar gait, the separation of the feet, the exaggerated lumbo-sacral curve, and the bulk of the calves are generally sufficient to distinguish Duchenne's paralysis from other conditions which are characterised by retarded development of locomotory power in early life.

You might be easily led to suppose, from the intellectual dulness which has marked our patients here, that the seat of the primary lesion in these cases is in the brain. This view was at one time held, but post-mortem investigation has hitherto failed to support it. In two cases in which post-mortem examinations have been made, eminent observers have failed to find any appreciable lesion of the nervous centres. The first case was reported by Eulenburg and Cohnheim, and the second by Bergeron and Charcot. In the cases before us,

the ophthalmoscopic examinations gave only negative results.

The remarkable mottling of the surface of the lower limbs observed in the cases I have narrated, may be referred to some peculiarity in the capillary circulation. The discolouration chiefly affected the lower limbs, but sometimes also extended to the upper extremities, but was never seen on the face or neck. It varied at different times from a bright rosy to a dusky red tint. Similar appearances have been noticed by Schutzenberger,[8] Berend,[9] and Griesinger.[10] In Berend's case the mottling was of a dusky blue colour, and was associated with a lowered surface temperature of the thighs. Griesinger states that in his patient the lower limbs were nearly always of an unnatural colour, sometimes rosy, at others dusky red. With this condition there was an elevation of the temperature of the surface above that of the trunk. Our observations made on the boys when in the hospital sometimes showed the temperature of the thighs to be a little above the normal standard, but there was no constant elevation or depression of temperature in any of our cases. The same observer (Griesinger) remarked that the discolouration became more pronounced

(8) *Gazette Médicale de Strasbourg*, 1862, No. 5.
(9) *Berliner Allgemeine Medizinische Centralzeitung*, 1863, No. 9.
(10) *Op. cit.*

when the patient made attempts to produce movements of the lower limbs, which he could not effect. A similar peculiarity I have frequently noticed in the patient W. M. (Case I): a kind of blushing of the limbs at their own powerlessness.

The prevailing hypothesis at present is that this condition indicates some lesion of the vaso-motor system as the cause of the disease. That long continued congestion is generally followed by the excessive development of connective tissue is a law, which has much evidence in its favour, and the sclerosis of the muscles in these cases may be another example of its action. The disease is probably due to some lesion of the vaso-motor system, but is not necessarily effected by means of the connective tissue hypertrophy. The other muscles of the body we have seen, become weakened as well as those which have increased in bulk. Some, indeed, never show any increase in size and yet become powerless, while the muscles most enlarged retain their strength longest.

I had at one time an idea that these cases might possibly be another manifestation of hereditary syphilis, and consequently a careful examination of all the patients and their families was made. The teeth of the boy whose case I have read were irregular, and the central incisors slightly serrated, but by no means notched. In only one case has a fairly characteristic central incisor been seen. In

none of the three families to which the children belong has a history of syphilis been found, and in none of the brothers and sisters of either of the patients have syphilitic teeth or traces of keratitis been discovered. In none of the cases have the children been the offspring of a consanguineous marriage, as has been observed in some cases in Germany. As far as our present observation goes, the disease is confined to children. It may occur in infancy, or it may appear as late as the thirteenth year.

There can be no doubt of the hereditary character of the malady in some cases. The boy whose case we first considered to-day comes of a healthy family; but the boy whose case is No. III, and who was in the hospital some months ago, was the third of his family affected. In this instance the two other brothers became completely paralysed, and were carried off by intercurrent diseases at sixteen and seventeen years of age. According to the mother's statement two of her brothers were similarly affected, and died when sixteen and seventeen years of age. It would appear from the history that one of her mother's brothers (the patient's great uncle) was affected with a similar form of paralysis. This would make a total of five cases in three generations. The brother of the patient, Zachariah W. (Case II), seems to have been a less well marked example of

this disease, in which there was no stage of muscular enlargement; this opinion is strongly confirmed by the microscopic appearances of the muscles. The girls of all these families have escaped, and continental experience points to the much greater frequency of the disease in boys.

Next as to prognosis and treatment. Paralysis with apparent muscular hypertrophy, when once fully developed, tends surely and steadily to a fatal termination. All the evidence hitherto collected confirms this. There is, however, according to Duchenne, a saving clause to a complete admission of powerlessness on our part. He has succeeded in curing two cases in the first stage by Faradisation of the muscles, which acts most probably by rendering the capillary circulation more active, and lessening the venous stasis, that favours the production of connective tissue. Our patient, admitted, as he was, at an advanced period of the second stage, was treated with iodide of potassium, iron, and cod-liver oil. He improved for a few weeks after admisson, probably more on account of the improved diet than the drugs. The daily application of the Faradic current was afterwards tried, but no good results were obtained, and when discharged the boy had certainly not improved. In the last stage of the disease Faradisation and Voltaism, however or wherever applied, appeared to have no beneficial effect.

When the pathogeny of the disease is more advanced our therapeutics will be more perfect. Careful observation will achieve this, and each of you may assist in the work.

POSTSCRIPT.

Case I. Sequel.—W. M. again came into the hospital under my care towards the close of 1873. From his father's account it appeared that after the boy's discharge in April, 1869, his power of walking had progressively decreased, and that in the course of eighteen months he had been reduced to crawling about the house. For some months before his admission he had been unable even to crawl, and had been obliged to remain in any spot on which he was placed. His father carried him down from his bed in the morning, propped him up in a chair or on a sofa, and there the lad remained all day.

When admitted, his wasted limbs and general powerlessness presented a marked contrast (Fig. 3) to the condition in which he had left the hospital in 1869. (*Vide* Figs. 1 and 2, p. 163.) In the interval he had grown in length of limb and size of trunk, his face was still well nourished, and his general intelligence was greater than formerly;

as far as muscular power was concerned he was, however, almost an inert mass, being able only to move his trunk sideways in the slightest degree by using his right arm as a lever.

When stripped his skin was observed to be harsh, dry, and more mottled than formerly; this

(Fig. 3.)

peculiarity had originally been confined to the lower extremities, but now also affected the surface of the trunk and upper extremities, and became much more distinct when he tried to move his limbs. When the boy was raised and supported in the erect position by two persons, one holding him under each axilla, and the other supporting him

from below by a hand placed under each tuberosity of the ischium, he could make no effort to rest his weight on his lower limbs; his legs, indeed, remained semi-flexed, the toes pointing to the ground and the heels being drawn up by the muscles of the calves, while the hamstrings prevented any straightening of the lower legs even by force. When held in position the spine was curved with the convexity backwards, and the former lumbo-sacral concavity could not be restored by any effort of the patient, even with the assistance of those who supported him. There was also noticed a slight lateral curvature of the spine to the left. When placed in bed the patient was able with a little support to maintain the sitting posture, but his position was most grotesque. He sat with his legs folded up in an exaggerated tailor-fashion, the heel of the right foot tucked up close under the pubic arch, while the concave plantar surface of the left foot was applied to the dorsum of the right. This was his favourite position. In order to gain it, however, he was obliged to use his hands to drag up his feet. When asleep he lay on his left side with his legs and thighs both drawn up, his heels touching his buttocks.

On further examination, the lower extremities were found to have diminished in bulk since 1869. The right calf measured only $9\frac{1}{2}$, the left $9\frac{1}{4}$, as compared with the former circumfer-

ence of 11 inches each. The thighs had lost less in circumference, the right measuring 12 and the left 11½ inches, or respectively ½ and 1 inch less than in 1869. The gastrocnemii muscles were fairly firm, but not so hard as formerly; they kept the feet in a state of equinism which could not be reduced. These calf muscles allowed the boy to make no movement beyond a slight extension of the foot, and a little flexion of the toes. The hamstring muscles, which were moderately large, retained the leg flexed upon the thigh. The glutei were still firm, but not hard and massive as formerly, and the lumbar muscles had also lost bulk and hardness to a similar degree. The muscles of the trunk were all much wasted. The abdomen was large and prominent, the liver was enlarged, extended three fingers' breadth below the costal arch, and caused a bulging of the right hypochondriac and epigastric regions. The organ was not tender, but smooth and elastic to the touch. The spleen was not enlarged. The chest was asymmetrical, the right side being the more capacious; the sternum was prominent, and directed towards the left, and produced a bulging of the cardiac region.

The respiratory movements were feeble everywhere, and especially so on the left side. The breath sounds were healthy all over the chest. The area of cardiac dulness was increased later-

ally, and extended from a finger's breadth beyond the right edge of the sternum to half an inch beyond the left nipple line. The upper margin of dulness was at the third rib, and the apex beat at the sixth rib. A loud mitral systolic murmur was heard all over the præcordial region, was loudest half an inch above the apex beat, and was distinctly heard in the axilla and on the left side of the spine posteriorly. This murmur was not present when the boy was in hospital in 1869.

The muscles of the upper extremities were much wasted, and had become so feeble that he could not raise his hands to his mouth, except when his elbows rested on the bed or table. In this position he managed to feed himself. The right could be raised more than the left, and was less wasted. The right deltoid retained a moderate amount of its fibres, while the left muscle had almost completely disappeared. The hands, thin and wasted, had scarcely any power of grasping, and could only be used to play with small light toys. The right arm at its points of maximum circumference measured only $5\frac{1}{4}$, the fore-arm $5\frac{1}{2}$ inches; the left arm was slightly less than its fellow, and had less muscular power.

In comparing the lad's state as described above with that which he presented formerly, it must be borne in mind that nearly five years had elapsed,

and that instead of a child of nine, the patient had become a lad of fourteen years. This no doubt accounts for the intellectual improvement exhibited. The heart-mischief which he had acquired in the interval was not referred by the parents to any distinct illness. He suffered from palpitation occasionally while in the hospital, and on one occasion had a sharp attack of congestion of the lungs with albuminuria, which, however, yielded to treatment. The digestive functions had remained good; occasionally he ate too much, and suffered in consequence: he had retained power over the sphincters.

The boy's hearing and sight had not suffered at all during the five years' interval, and his face had lost some of the stolid expression which had previously characterised it, although it still lacked intelligence and animation. Sensation to tickling by a hair, and to heat and cold was everywhere perfect. The mottling of the skin was very remarkable, and was possibly increased by the changes in the circulation consequent on the mitral insufficiency. The temperature of the surface of the lower extremities taken in the groins, in the popliteal spaces, and over the calves was generally normal: sometimes, however, it rose as high as 99° and at others fell as low as 97°; the temperature was highest when the mottling of the surface was of the reddest colour. The

asymmetry of the chest and the lateral curvature of the spine find an explanation in the influence of the weight of the viscera, especially the enlarged liver, on the cavities of the thorax and abdomen, which had no longer the support of healthy muscles. Sleeping as the boy did, always on the left side, the enlarged liver pressed towards the left, and so caused the asymmetry of the chest and the lateral curvature of the spine.

When admitted, the electric sensibility of his general surface to Voltaism was somewhat diminished; over the nerves it was increased, probably from the close proximity of the electrodes in consequence of the emaciation. The electro-muscular contractility was very small. To Faradisation the electric sensibility was unusually small. The electro-muscular contractility varied with the size of the muscles, being greatest where the muscles remained largest, namely, on the flexor surfaces of the body generally, on the right side, and in the lower extremities. The right deltoid muscle, especially in its anterior fibres, responded to the faradic current much better than the same muscle on the opposite side, which had almost entirely disappeared.

While the boy remained in the hospital, Faradisation and Voltaism were in turn tried, but had no appreciable effect in restoring muscular power. The

deltoid muscles were specially selected for observation, but in spite of the application of both forms of electricity, a month's trial of each form being made, there was no improvement in the power of raising the arm, and no increase in size of the affected muscles. The boy during a four months' stay improved in general nutrition, but, while he apparently lost no muscular power, he certainly gained none under the electrical treatment.

VIII.

OBSERVATIONS ON DIABETES MELLITUS AND ITS TREATMENT.

In a disease like Diabetes Mellitus, in which success seldom crowns our treatment, in spite of the very extensive knowledge we possess of the physiological mechanism of the disorder, it is, I think, necessary to have some hypotheses to give interest and method to clinical work. Hypotheses are unmitigated evils only when they cease to be the servants and become the masters of facts. Kept in their proper places, recognised only as provisional explanations, and not mistaken for proven truths, they favour instead of obstructing clinical research. It is with this view that I have ventured to arrange the cases narrated in this paper, according to certain hypotheses respecting the nature of the malady. If the causes of the glycosuria differ as widely as I suppose, the methods of treatment suitable to the different forms must vary as greatly. It is, I believe, the

absence of a clear recognition of the special variety of diabetes in each case, which has hitherto made our efforts to treat this malady so unsuccessful. Unable to separate the forms of the disease clinically, we have been obliged to treat all on common principles, and consequently the results drawn by different observers from different cases have been often most contradictory.

I.

Regarding the disease from a purely clinical point of view we observe great differences in the effects of diet. In some instances the withdrawal of all starchy food from the dietary will cause the fundamental symptom, the glycosuria, to disappear. Patients of this kind are often gouty, and, in spite of their diabetes, remain well nourished and capable in many instances of leading comparatively active lives. This form of diabetes seems to me to depend not on a disorder of the gastric and intestinal digestion, as some have supposed, but rather on a defect in the power of the liver to assimilate and store up the sugar derived from starchy food. In health, the sugar thus formed when it reaches the liver is fixed in the liver cells as glycogen, and it is this process of fixing or hiding away the sugar —glycocrypsis we may call it—which seems to

be at fault in this class of patients. The raw sugar formed from starch passes through the liver and into the general circulation, and is excreted by the kidneys; for Bernard has shown that the sugar thus formed is not oxidised in the blood as true liver-sugar is. The glycosuria, therefore, disappears when these patients abstain from starchy food. Andral's oft quoted case, in which glycosuria was present, although the vena-porta was obliterated, finds its explanation in the fact that the sugar entered the general circulation instead of being assimilated by passing through the liver.

In other and more numerous cases of diabetes the glycosuria is only partially checked by a purely animal diet, the liver continuing to form sugar from the albuminous articles of food. The normal function of the organ is over-active, and an excessive quantity of sugar is produced; the amount of this over-production on animal food is a good test of the gravity of the case. In diabetics of this class the bodily temperature, as in the former class, is maintained at the normal standard at all events during the earlier period of the malady, and they remain for a time well nourished, and may even grow fat (*vide* Case III). The enormous appetite of such patients more than balances the waste by glycosuria; when, however, the digestion fails, they break down quickly.

In these, and also in the next class of cases, a prolonged use of, and a too sudden transition to, purely animal food often proves injurious, and in my opinion hastens the development of the pulmonary complications. The man whose case forms the third of those that follow was a remarkable example of the improved nutrition following the return to mixed diet, and he laid up in his tissues a large amount of reserve fuel in the shape of fat which no doubt supported him for some time, when the digestive organs were breaking down under the prolonged strain. In some milder cases of this kind the body fails to use up all the sugar formed: there is a relative rather than an absolute excess of sugar. Under such circumstances, by altering the conditions of the patient's life, and especially by increasing the amount of muscular action (*e. g.*, by exercise), the slight relative excess of sugar may be consumed by the muscles, and the ill effects of the disease avoided. In other cases, the sugar is possibly in relative excess on account of a deficiency of the ferment which normally converts it in the muscles into lactic acid and glycerine, and so fits it for combustion.[1]

(1) A very carefully compiled summary of our knowledge of the various causes of glycosuria is given by Dr. Brunton in his "Lectures on the Pathology and Treatment of Diabetes Mellitus," in the *British Medical Journal* for January and February, 1874. Dr. Brunton's Lectures form so valuable and complete an exposition of our experimental

There is again, I think, a third class of cases in which the restricted diet limits the amount of loss of sugar by the urine, but has no power of making it cease. These cases are less satisfactorily nourished than those in which the sugar formation is increased, and they have nearly always a temperature lower than normal, it may be one or several degrees. In these cases we can hardly explain the low temperature on the hypothesis of diminished combustion of sugar by oxidation, seeing that there is plenty of sugar to be oxidised, and seeing also that the excessive daily loss of urea proves that the oxidation processes are still most active. If ordinary liver sugar were formed in excess it ought to save the albuminous tissues, and yet these patients waste like fever patients. To explain such facts we can, I think, find no better hypothesis than that of Cantani, which assumes the production of an abnormal liver-sugar. Bernard has proved by injections into the veins and tissues of animals, that the sugar formed by the liver in health is used up in the circulation of an animal and does not appear in the urine, while diabetic sugar is less perfectly destroyed. These experiments point to a differ-

knowledge of glycosuria, that after reading them I have omitted much that I had written for the first part of this article, and would refer my readers for a full statement of the whole subject and the authorities to his Lectures.

ence between the two sugars, a difference which escapes chemical or ordinary physical analysis, but which the living body detects. The sugar produced in this form of diabetes we may regard as an altered liver product, a defective secretion, which the liver produces in larger quantities probably than in health, and not only excessive in quantity but deteriorated in quality, an association which is common in all products of gland action. This diabetic sugar no longer consumable in the economy does not save, by its oxidation, the fat and albuminous tissues. At first probably formed in small quantity (for there is no reason to assume, as is generally done, that all the liver cells are affected at once), it appears in small quantities in the urine, and an increased consumption of food makes up for the loss. Gradually, however, the process spreads, and although increased activity of other parts of the liver, aided by excessive appetite, may for a time keep up the nutrition, yet the strain is too great to last long, and sooner or later the glycogenic function is wholly disordered and nothing but diabetic sugar is formed. The fats of the body, and later on the albuminous tissues, are consumed by the oxygen, and the patient steadily wastes. We have, in short, a profound modification of healthy nutrition, of which the chief feature seems to be an excessive oxidation of the albuminates and fats.

These, no longer protected by the production of healthy liver sugar, are consumed in its stead in order to maintain the necessary animal heat. To this state, I think, cases of the second class beginning with excessive sugar production eventually come: the deterioration in the quality of the sugar following on the over-production. In this way we can account for the rapid wasting and large urea-excretion which mark the advanced stage of cases that undoubtedly began as cases of superabundant sugar formation.

The three hypotheses which I have sketched above appear to me to be justified by the present state of our knowledge. I advance them only as provisional explanations of the disease. They are:—

1. Defective power in the liver of assimilating and storing up the sugar formed from starch during digestion. This I propose to call diabetes from defective glycocrypsis.
2. Increased production of sugar in the liver or diabetes from excessive glycogenesis.
3. Abnormal formation of sugar in the liver as regards quality (paraglucose of Cantani) or diabetes from abnormal glycogenesis.

I have arranged the cases which follow under these several classes. In attempting this difficult task, I have been guided by the clinical features

presented by the patients and by the effects of remedies. In some instances, especially in the advanced stage of the second and third forms of the disease, it is sometimes difficult, if not impossible, to say to which form a particular case may belong. In the remarks at the beginning of each section, however, will be found a brief reference to the special characters of each set of cases.

II.

Before proceeding to the narration of the cases, I have thought it well to indicate the manner in which they were treated, as regards the diet given and the drugs administered.

The amount of sugar lost under ordinary mixed diet and under restricted diet containing little or no saccharine or farinaceous elements has been estimated in most of these cases, and in the few in which it could not be done the results are, nevertheless, trustworthy. This preliminary inquiry as to the effects of diet is important, not only to enable us to recognise the form of disease we have to treat, but also as a safeguard against the fallacy of referring to the action of a drug an improvement which is really the result of a modified diet. Instead of giving daily records of the quantity and quality of the urinary secretion, I have recorded the results in the form

of the *average* daily excretion for the period of each kind of treatment. This has been done for the sake of brevity, and also because diagrams have been made from time to time of the progress of each patient; and it is difficult to illustrate in a diagram of convenient size the daily alterations in the quantity of water and sugar for a period of several months. I am aware that this method of recording the average daily excretion is open to some objections, the chief of which, however, is that it represents the effect of treatment in a much less striking way than a daily record which shows the progressive diminution of sugar and urine. Daily analyses of the urine were made in nearly every case, and in the exceptions they were made three or four times weekly. In all cases the urine of twenty-four hours was measured into a single vessel, and the specimen taken thence was examined for its specific gravity and sugar contents.

The following drugs were given, and after each drug are stated the conclusions which the study of the cases seems to justify.

The ethereal solution of peroxide of hydrogen was given to two patients. It had little or no influence in diminishing the sugar excretion in Case IV, an example of over-production of sugar. In Case XIII, an example of abnormal glycogenesis, the sugar and water fell, the skin acted most

copiously, and the perspiration contained a large quantity of sugar. The improvement in the state of the urine was, no doubt, partly due to this second channel of elimination, but the improvement was too marked to be wholly accounted for in this way. I am inclined to attribute the beneficial effects in this and in other cases, to the ether, rather than to the peroxide of hydrogen. The ether, by stimulating the pancreas to increased secretion, insured the more perfect transformation of starchy food in the intestine, and thus, in place of sugar, lactic acid was produced, which after absorption was burnt off, increasing the temperature of the patient, promoting perspiration, and saving the albuminous tissues. It is possible that the increase of pancreatic secretion produced by the ether may influence the glycosuria in some cases in another way. The pancreatic fluid contains a large amount of ferment, and this ferment when formed in increased quantity by the gland may be absorbed from the intestine, and after absorption may aid in transforming any excess of sugar in the blood into lactic acid, and so render it fit for combustion. This suggestion obtains support from the well-known fact that many observers have found a degenerated state of the pancreas associated with glycosuria.

The liquid extract of ergot was given in four

cases of excessive glycogenesis, one of which was complicated by extensive lung and kidney mischief. The ergot was given on the hypothesis that it might lessen the congestion of the abdominal viscera. The effects observed to follow the use of ergot were—(1) decrease in the quantity of urinary water, (2) slight, but in one case considerable decrease in the quantity of sugar, (3) slight fall in the specific gravity of the urine. The decrease in urinary water I have also observed in cases of diabetes insipidus, as well as in other cases of true diabetes.

The bicarbonate and citrate of potash were given in succession to the same patient—whose case was one of excessive glycogenesis. They were given on Mialhe's notion, that by correcting the supposed hyper-acid state of the blood they would aid the decomposition and combustion of the sugar. The results show that they have no power of diminishing the quantity of urine or the amount of sugar. In other cases I have obtained similar results with alkalies. It is worthy of note that in no case was the acid reaction of the urine neutralised, although the urine is twice noted as being only faintly acid.

Opium was given to the same patient after the potash salts. The quantity of water fell, the specific gravity remained very constant, and the quantity of sugar was lessened. The patient,

however, improved manifestly out of proportion to the diminution of the sugar—an improvement which I have noticed in other cases under opium, when the sugar diminution has not been great, and which I am inclined to attribute to the influence of the opium in lessening the urea excreted. In the case here recorded the urea fell, in the course of ten days nearly 120 grains. The opium, however, did not act nearly as well as it does sometimes. It was also given in small doses with beneficial results to the two cases of defective glycocrypsis.

Codeia was given to one patient to the extent of four grains daily, after lactic acid had been used. It produced in these doses vertigo and unpleasant head symptoms, and had to be discontinued. The sugar and water fell considerably under its use.

Bromide of potassium in combination with Tr. Ferri Perchloridi was given in Cases X and XIV. This combination I have found much more useful in diabetes than the bromide alone; I have, however, only found it beneficial in mild cases. In such cases it has some slight influence in diminishing the amount of water and checking the waste of the albuminous tissues; under its use the quantity of sugar fell and the appetite was lessened. The Tr. Ferri Perchloridi was given alone in one case of excessive glycogenesis. The

patient (Case III) improved greatly in nutrition while under this treatment, but continued to lose large quantities of sugar.

Lactic acid was specially tried in several examples of excessive glycogenesis; in three of these cases the lactic acid was given in small doses, not exceeding two drachms a day. In Case IX well-marked rheumatic symptoms were produced, the quantity of urine fell, the specific gravity was not much affected, the sugar was diminished in quantity, and the patient improved. In Case X less marked signs of rheumatism occurred. In a third case the sugar was not estimated, but the quantity of urine was lessened, and the specific gravity fell slightly, while no rheumatic symptoms appeared. In a fourth case the lactic acid was given in much larger doses: to the extent of 3 oz. daily. In this patient no rheumatic symptoms whatever were produced. The urinary water was diminished, and the sugar fell slightly, rising again when the acid was discontinued. The functions of the skin were generally restored by the lactic acid, and in this way the decrease of water may be wholly or partly explained. Thirst was not particularly diminished, and the appetite was, if anything, increased. From these cases it appears that lactic acid has little or no direct influence on the glycosuria in diabetes from excessive glycogenesis.

It is in diabetes from abnormal glycogenesis that lactic acid is especially indicated. In this form of the disease the sugar produced by the liver is no longer capable of undergoing the normal conversion, whereby the heat of the body is maintained. To meet this condition therapeutically, Cantani pointed out that some combustible substance must be administered to support the temperature of the body, while the most complete rest possible is given to the liver by the use of a purely animal diet. He selected lactic acid as the best combustible agent, and his treatment consisted in the use of this drug, and strict diet. Physiological experiment has shown that sugar is converted into glycerine and lactic acid before combustion, so that by giving either of these drugs we supply the very matter which is wanting in the system in cases of abnormal glycogenesis. The treatment by skim milk, which is an approach to this method, owes its value to the lactic acid formed from the lactin of the milk, and to the fact that the patient on this diet has, in addition to his lactic acid, a purely animal food. The same may be said of the use of koumiss, and of the sour-milk and butter-milk cures which have from time to time been recommended.

In Case XII are seen the good effects of glycerine, while Cases XIV and XVI illustrate the beneficial action of the skim milk treatment.

The use of lactic acid in addition to a diet of skim milk is illustrated by Case XV. In mild forms of diabetes this treatment and that by skim milk alone have often succeeded in my hands.

In one case (XVI) of abnormal glycogenesis other drugs were tried—viz., *Arsenic* and *Valerian*. I have sometimes seen arsenic act well, by improving the nutrition of the patient and lessening the thirst. The sugar has not in my experience fallen much under the use of arsenic, and I am therefore inclined to consider that it acts mainly by saving the waste of albuminous tissues. It is in this way that valerian, especially the extract, appears to me to be useful. It lessens the amount of water and diminishes the sugar slightly, but the patients seem to improve out of all proportion to the diminution of sugar. The beneficial action of valerian is to be referred to its effect in diminishing the quantity of urea excreted. This is its first effect, the diminution of the water follows, and the glycosuria appears to be affected later, as Bouchard has pointed out.[2]

(2) *Medical Record*, Dec. 24, 1873.

III.

CLASS I.—DIABETES FROM DEFECTIVE GLYCOCRYPSIS.

Cases which come under this head are commonly called starchy diabetes, and are simply examples of glycosuria, depending on an abnormality in the evolution of starchy food. The sugar comes wholly from the starch and sugar of the diet not being arrested and stored up in the liver cells as glycogen. The first case below illustrates this very clearly, and the second also shows the influence of purely animal food in causing the disappearance of the sugar from the urine. Cases of this kind often end by becoming permanently diabetic, for as Bernard has shown, a faulty condition of nutrition, such as these patients suffer on account of the loss of their starchy food, is followed by an increased activity of the glycogenic function of the liver. This over-production of sugar from albuminous articles of food may become permanent, and the case then falls into the second class of increased glycogenesis. The patient, M. S. (Case II), has, since I last saw her, I am informed, become much worse; and has most probably become an example of excessive glycogenesis, especially as there were indications of

this morbid course in the inability of a purely animal diet to completely check the glycosuria during the last few weeks she was under my care.

In the treatment of this form of diabetes a judicious restriction of diet is the most important point. Drugs are not of so much value as in the other forms; opium, however, in small doses is very useful in checking the secondary development of excessive glycogenesis. Stimulants in small quantities are sometimes useful, and the ethereal solution of peroxide of hydrogen has in my experience occasionally been taken with benefit. As cases of this class do not serve to illustrate the effects of remedies on the sugar excretion, I have only appended two examples of diabetes from defective glycocrypsis.

Case I.—*Treated by diet and small doses of Opium.*

John C., a jeweller, æt 44, came into hospital suffering from weakness and frequent micturition. He was a small man, only moderately well nourished, and before admission had undergone a good deal of privation from being out of work. He referred the first appearance of his symptoms to a date some fifteen months before he came to the hospital, and said he had suffered ever since from thirst, large appetite, frequent and copious mictur-

ition, constipation, and a dry harsh state of the skin. At first he had a good deal of indigestion, and was more thirsty than he has been latterly. He never had any serious illness before the present attack; always lived well and drank freely, and was rather stout and well nourished up to the time of the aggravation of his symptoms through deficient nourishment while out of work. At the time he was admitted he had lost flesh considerably, but was not greatly emaciated. The skin was dry and dusky. The temperature 98° F. The tongue reddish, dry, and only slightly furred. The teeth were nearly all gone. His appetite was large, and thirst considerable, but not so great as formerly. The pulse was feeble; the heart-sounds were clear, but the impulse was weak. The lungs were healthy. The liver and other abdominal organs presented no signs of disease. The urine was pale and copious—90 oz. a day, specific gravity 1050, and contained 30 grs. of sugar in the oz., or at the rate of a total daily excretion of 2,700 grs.

As the patient had been living chiefly on starchy food before admission, the change to ordinary diet in the hospital, with a free supply of meat, produced a marked effect in improving his appearance and restoring his strength. The effects of this diet on the glycosuria, and the results of other treatment are stated in the following table:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
98	1048	28	2744	Average of 4 days on mixed diet, with a free supply of meat and milk.
67	1036	15	1005	Average of 4 days on diet composed of meat, bran bread, milk, green vegetables.
64	1027	7	448	Three days, on which same diet as above was taken with this difference, that the 1st day only ⅓ the quantity of bran bread was allowed, and on the other days none was taken.
60	1028	5·6	336	
52	1026	3·5	182	
48	1025	Trace		Four days on purely animal diet consisting of meat, eggs, and skim milk.
44	1023	„		
40	1017	None		
32	1019	„		

After this the man rebelled against the purely animal diet, preferring to take the risk of a partially restricted diet. On this, with the use of three grains of opium daily, he improved greatly, although the sugar reappeared and reached on three occasions as much as 12, 14, and 15 grs. in the oz. A diminution of the quantity of bread in his food caused it to fall again. He gained flesh and strength and left the hospital six weeks after his admission, having gained 9 lbs. in weight.

Case II.—*Treated by diet, Lactic Acid, and small doses of Opium.*

Mrs. M. S., æt 59, a widow, came from the country to consult me for diabetes, from which she had suffered for some fourteen months. She

referred her illness to the fatigue she underwent while nursing her husband during a tedious illness, and to the grief occasioned by his death. Soon after that event she began to feel weakness, and to be troubled by great thirst, polyuria, and increased appetite. After a month or two she began to lose flesh, and from being fairly plump had become thin. She was a small woman, slight but not emaciated, weighing 108 lbs., with a dry sallow skin and anxious expression. She had suffered from gouty symptoms before her present illness. Her tongue was furred, dryish, and red at the edges. The heart's sounds were healthy, and there was nothing abnormal in her lungs. The liver was not enlarged, but there was some tenderness in the right hypochondriac region. The spleen was normal in size. The bowels were regular. She stated that she was passing about eight pints of water daily. The specimen brought had a specific gravity of 1036, and contained 27 grs. of sugar in the ounce, representing a total daily excretion of 4,320 grs. She was ordered a diet composed of under-done or raw beef and mutton, skim milk, bran bread (made according to Camplin's formula), and green vegetables. I prescribed a drink containing 60 minims of lactic acid in 10 ounces of water, to be taken during the day, and a grain of opium at bed time. As the patient lived at a distance I did not see her for

three weeks, when she stated that she was much better; the urine had greatly decreased in quantity, at the end of the first week of treatment was only 4½ pints a day with a specific gravity of 1027, and had since fallen to 3 pints with a specific gravity of 1022, 1018, and 1020 on the three days preceding her visit. This urine was examined for sugar, and found to contain only a trace. A week later she returned, her water having increased to five pints with a specific gravity of 1027. This change was due to the use of a freer diet in which she had indulged, thinking herself safe. The water was found to contain 7.5 grs. of sugar in the ounce, and represented a daily loss of 750 grs. She was ordered to return to her restricted diet, and to take a grain of opium three times a day. As the thirst was much less she had some time since discontinued the lactic acid drink. Under this treatment the water fell during the next week to 3½ pints, but as the specific gravity was 1028, following my directions she omitted the bran bread from her diet and came to me a week afterwards with the urinary secretion reduced to 54 oz., with a specific gravity of 1020, and containing in one of the specimens brought a trace and in the other no trace of sugar. While on this purely animal diet a deposit of uric acid frequently appeared in the urine. She continued the diet for a week

longer, and then took to the bran bread again. When I last saw her, three weeks later, she had gained 5 lbs. in weight, and was passing only from 50 to 60 oz. of water which contained but a trace of sugar.

CLASS II.—DIABETES FROM EXCESSIVE GLYCOGENESIS.

The cases under this head serve not only to illustrate the special features of this form of the disease, but are of interest as showing the effects of certain drugs on the chief phenomena of the malady. They present a strong contrast to the two first cases in their behaviour under restricted diet, sugar being still formed by the over-active liver, even when all starch is removed from the food. The albumen supplies the material which the liver in these cases transforms into sugar. This process of excessive sugar formation may go on without producing emaciation; it may even be associated with increased nutrition, as in Case III, so long as the digestive organs are active enough to supply sufficient assimilable material to maintain the necessary surplus over and above the sugar loss. Cases V and VI illustrate the period when the balance is maintained with difficulty, and emaciation is beginning, but can still be arrested by judicious diet and treatment. Cases IV and VII

are examples of the later period when the digestive powers fail to meet the enormous sugar loss, and the rapid emaciation indicates the profound nutritive disorder which marks the last stage of the disease. The four last cases (VIII, IX, X, and XI) were under my care about the same time, and were all treated with lactic acid. The effects of the treatment are given in the tables. Of these cases No. X was not a well-marked case of excessive glycogenesis, but was, I am inclined to believe, rather an instance of relative excess of sugar than of over-production. The comparatively small amount of sugar excreted, and the great diminution which took place under restricted diet, and plenty of open air exercise, favour this view. Of this form of glycosuria, and of that form depending on a deficiency in the ferment which normally converts the sugar into lactic acid in the muscles, we have as yet no knowledge clinically, and I have therefore thought it better to group all the cases under the head of Excessive Glycogenesis. The post-mortem appearances in Case VII are worthy of study, as affording post-mortem evidence of the increased vascularity of the liver and all the portal system. Although the post-mortem examination failed to discover any nervous lesion in this case, the appearances correspond so closely with those which Cyon and others have observed to follow section of the splanchnic nerves,

and of the nervous ring (annulus of Vieussens) round the subclavian artery, that we are justified in regarding this case as an example of glycogenesis, similar in its mechanism to the artificial forms which physiologists have experimentally produced.

In the treatment of this form of diabetes it is necessary to combine a carefully regulated diet with the administration of some drug which directly controls the activity of the hepatic circulation, and so checks the excessive glycogenesis. Opium acts mainly in this way, and so does its derivative Codeia. My recent experience strongly confirms Dr. Pavy's high estimate of the value of Codeia. In a minor degree Ergot is valuable, and a combination of the drugs deserves a trial. The other remedies used in the following cases are shown to have had little or no influence on the glycosuria.

Case III.—*Treated by diet and Tr. Ferri Perchlor.*

A. D., æt 24, iron caster, a short man, of florid complexion and somewhat spare habit, was admitted into hospital suffering from diabetes. His illness dated from some four months prior to his admission, and began with pains in the legs after work and a sensation of dull pain and weakness in the lumbar region. With these symptoms, ex-

cessive thirst, inordinate appetite, uneasiness in the stomach, flatulence after meals, and frequent and copious micturition were soon associated. When first seen in hospital he complained of all these symptoms; his skin was dry and branny; his gums were red and tender; his tongue was furred but raw and red at the edges; the bowels were costive; the heart and lungs were free from disease; the abdomen was rather full at the upper part, and the liver enlarged, reaching about $1\frac{1}{4}$ inches below the costal arch. Slight pain was felt on pressure over the kidneys. His weight was $120\frac{1}{4}$ lbs., but before this illness it had been 133 lbs. The wasting had chiefly occurred during the past month while he was unable to work, and had consequently been less well-fed than previously.

The muscles were still fairly well-developed, and the muscular power was not greatly diminished. He felt, however, an unusual sense of fatigue and muscular pain after any exertion. The urine had a specific gravity of 1043, and contained a large quantity of sugar. He was first placed on a diet consisting of mutton, bread, milk, beef tea, and potatoes, and the average daily excretion of water and sugar on this and other diet was recorded as follows:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
210	1043	38·1	8001	Average of 5 days on mixed diet; Tr. Ferri Perchlor, ʒss in day.
176	1042	37	6512	Average of 10 days on diet composed of meat, milk, beef tea, bran bread, and cabbage.
160	1043	38·5	6160	Average of 6 days, same diet except almond bread instead of bran bread.
168	1046	47	7896	Average of 1 week; the restricted diet was now discontinued, as the patient expressed his determination to go out rather than continue it; the same diet was ordered as on admission, bran bread only being substituted for white bread.
152	1035	32·5	4940	Average of last 14 days spent in hospital, diet as above; treatment Tr. Ferri Perchlor ʒss in day; the man improved in appearance and gained flesh on his mixed diet, weighing at time of discharge 128 lbs., a gain of more than 7 lbs.

I met this man in the street more than two years after he left the hospital, looking stout and well. He had increased greatly in flesh, having gained during the two years no less than 27 lbs. He had been able to work and had lived freely, drinking two or three pints of beer a day and eating plenty of meat, and occasionally brown bread, but not many potatoes. He came up to the hospital at my request, and on examination his chest was found to be healthy; the tongue was red, raw, and fissured; the skin dry, thirst excessive, and the appetite very large. His sight was good. There

was slight œdema about the ankles which he said was worse after extra work. He still passed urine in large quantity: his estimate was 12 pints in the 24 hours, a specimen which was examined had a specific gravity of 1038, and contained 37 grs. of sugar in the oz. Soon after this his digestive powers began to fail him and he broke down some six months after, dying quickly from lung complication. I did not see him after the date at which his urine was last examined at the hospital, but gathered the particulars of his death many months after its occurrence from my friend Mr. Hollinshead, of Selly Oak.

Case IV.—*Treated with* (1) *Ext. Ergotæ Liq. and* (2) *Peroxide of Hydrogen.*

Elizabeth J—, æt. 40, governess, florid complexion, sandy hair. Her family history is good; she always enjoyed good health till some twelve months ago, when she was about to be married. A week before the wedding day her intended husband was thrown from his horse and killed. She was severely affected by the accident, and for some weeks confined to her bed. On getting about again she noticed that her strength did not return, and that she suffered from obscure pains in the back and in the head. About three months later she noticed that she passed an unusual quantity of water. Her appetite was good

and thirst great. She lost flesh, and at the time of her admission to the hospital weighed only 100 lbs., having lost 21 lbs. The skin was dry; the lowest temperature observed was 97.6°; the tongue was red and fissured, the expression anxious, the temper irritable. The heart and lungs were free from disease, the appetite was voracious, and the bowels were confined. After being under treatment for nine weeks she left the hospital, having gained 2 lbs. in weight. She died 12 months afterwards of diabetic phthisis. On admission she was placed on ordinary diet for four days. The report of urine was as follows:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
185	1032	25·5	4717·5	Average of 4 days on ordinary mixed diet.
160	1030	24	3840	Average of 4 days on partially restricted diet, *i. e.* meat, milk, green vegetables, and bran bread.
135	1027	23·7	3199·5	Average of 10 days on same diet; Liq. Ergotæ ℥iss in day.
170	1030	19	3230	Average of 8 days on same diet; no ergot.
116	1027	16·25	1885	Average of 5 days on same diet; Liq. Ergotæ ℥iij a day.

At this period the ergot was stopped on account of pains in the lower limbs, and general malaise, the water increased the next day to 130 oz., but the sugar fell for two days till it reached only 11 grs. per oz. Peroxide of hydrogen was

now given to the extent of five drachms daily. Under its use, and with the restricted diet previously taken, the urine averaged for the first four days 120 oz., and the sugar excretion 2,400 grs. On mixed diet the sugar rose to a daily average of 4,633 grs., and the water to 156 oz., in spite of the peroxide. The last seven days on restricted diet and this drug gave a daily average of 126 oz. of urine, and a daily loss of 2595·6 grs. of sugar. The peroxide thus failed to maintain the improvement produced by the ergot.

Case V.—*Treated with* (1) *Ext. Ergotæ Liq.*, (2) *Bicarbonate of Potash*, (3) *Citrate of Potash*, (4) *Opium.*

Alfred S—, æt. 22, miner, florid complexion, lightish brown hair. The family history was good. He was always strong and healthy till seventeen months before his admission into the hospital, when he had a bad attack of typhoid fever, and relapsed in consequence of getting up too soon. He never regained his strength, and always felt thirsty. During his convalescence he noticed that he passed a large quantity of water. He got thinner, in spite of a large appetite; the bowels were always troublesome, and the skin was very dry and rough. On admission his lungs and heart were found to be free from disease; the tongue was furred. Weight 120½ lbs. This man left the

hospital at the end of seven weeks, having improved a good deal under the opium treatment, which, however, could not be pushed beyond sixteen grains daily, on account of head symptoms. When he left the hospital he weighed 127½ lbs. He continued the opium treatment for some time, and improved considerably, passing generally under 100 ounces of water, with sometimes as little as twenty-one grains of sugar in the oz. I have lost the memorandum of the analyses made at this period, so have not been able to include results in the table.

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
220	1042	41	9020	Average of 3 days on ordinary mixed diet.
164	1038	35	5740	10 days average on restricted diet.
124·5	1042	41·8	5204·1	7 days average, restricted diet; ʒiiss Liq. Ergotæ daily.
134	1039	46·5	6231	7 days average, restricted diet; bicarbonate of potash ʒvj a day; urine acid, but on two days, only slightly so.
141	1038	44	6204	6 days average, restricted diet; ʒiss of citrate of potash daily; severe purging produced after 6th day, therefore medicine was stopped; urine never lost its acid reaction.
132	1038	47·1	6217·2	Average of 7 days, restricted diet; 5 grs. of opium a day.
118	1039	40·8	4814·4	Average of 7 days, restricted diet; 12 grs. of opium a day.
124	1039	42·2	5232·8	Average of 7 days, restricted diet, 6 oz. of white bread; 15 to 16 grs. of opium a day.

In this case the urea was estimated five times

during the last ten days of the opium treatment. On the first occasion it was 820·15 grains a day; then it fell as follows:—730·08 grains, 645·12 grains, 713·16 grains, 701·8 grains a day. The last two estimates were made while the patient was taking white bread extra.

Case VI.—*Treated with Ext. Ergotæ Liq.*

Sarah A. H—, æt 22, single, plate-glass smoother, florid complexion, dark hair. The family history was good. Her father, who suffered from chronic rheumatism, died about eighteen months ago. She was very greatly distressed at her father's death, and has never felt well since, being low-spirited, and not caring to mix with any of her friends or companions. The present illness began by pain in the loins, especially on the left side. When at work she was easily fatigued, and when tired the pain in the back increased, and pain in the head came on. She was very hungry and thirsty at this time, and drank freely of water and ginger beer. She passed water very frequently, and was much troubled by pruritus vulvæ. The menstrual functions were regular, but the discharge was scanty and like dirty water. About two months before admission she began to lose flesh rapidly; her fingers gathered about this time, and she lost several of her nails. She was treated at home

for two or three months, but applied at the hospital as she became worse. On admission (May 23rd, 1869) she weighed 115 lbs.; her thoracic organs were healthy; the liver was somewhat enlarged. There was tenderness on pressure over the left kidney region. The tongue was red, with pale fur at the base. The appetite was excessive, and the bowels were very obstinate. The skin was dry and branny. She passed 320 to 360 ounces of water in twenty-four hours. The reports of the urine and the results of the treatment were as follows:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
310	1040	50	15500	Average of 3 days on ordinary mixed diet; temperature 96·5° to 97·2°.
262	1043	48	12576	Average of 7 days, partially restricted diet, *i. e.* bran bread, meat, milk, green vegetables.
230	1044	49	11270	Average of 7 days, diet as before; Liq. Ergotæ ʒiss daily; temperature 97·5° to 98·4°.
180	1040	38	6840	Average of 7 days, diet as before; Liq. Ergotæ ʒiij daily; temperature 97·5° to 98·4°.

The ergot was now stopped, as the patient complained of great pains in the abdomen and legs, and general malaise. Soon afterwards she got restless and left the hospital, wishing to be with her friends. The case was a most severe one; it illustrates the effect of ergot in diminishing

the quantity of water. The fall in the amount of sugar was considerable, greater, I think, than the restriction of diet would account for. When discharged she weighed 118 lbs.

Case VII.—*Diabetes complicated with albuminuria and pneumonia; treated with Ergot.*

Jane F—, æt. 27, single, laundress. There was no family history of diabetes. She has had peritonitis, and six years ago variola; she has never been as strong as before the latter illness. About three years ago she began to pass an excessive quantity of water; her appetite was hearty, but not voracious; her thirst was great. Emaciation began soon, and she has lost flesh ever since. About twelve months before admission into the hospital she caught cold, her legs swelled, and she noticed that she was puffy under the eyes; she also had a distressing cough. She has been unable to work for some months, and has for some weeks been confined to her bed. When admitted (November 1st, 1870) she was greatly emaciated, the face was puffy, the legs œdematous, the tongue was moist and red, the bowels confined. The heart-sounds were healthy. The upper lobe of left lung was dull on percussion, both in front and behind. Under the middle third of left clavicle was detected a cavity with fluid contents; in its neighbourhood were heard

coarse mucous râles, and lower down bronchial respiration. The chest was flattened on left side. The right apex was dull on percussion, with harsh breathing, almost bronchial, and prolonged expiration. The liver was enlarged, projecting about an inch and a half below the costal arch. The urine amounted to 130 oz., in the 24 hours, sp. gr. 1040, and a *neutral reaction*; it contained a large quantity of sugar, about $\frac{1}{8}$th albumen and a few epithelial casts. Temperature 97·4°. She was placed upon milk diet, and ordered Ext. Ergotæ Liq. m. x ter die. During the next week the urine fell in quantity as follows:—110, 112, 60, 96, 106, 80, and contained from 35 to 46 grs. of sugar an oz., the specific gravity remaining nearly stationary. Her temperature fell as low as 95·4° on several occasions in the morning, but began, about November 9th, to rise as high as 98° in the evening. The softening in the left upper lobe went on extending, but in other respects she appeared better. On November 16th her morning temperature was as low as 94·4°. The urine was 90 oz., sp. gr. 1040, with 29·5 grs. of sugar an oz., or 2655 grs. in twenty-four hours. At this time she was taking ʒiss of ergot daily, and in addition to four pints of milk she consumed 8 oz. of bran bread, one egg, and a chop. In the evening of this day fine crepitation was detected at the left mamma, and the dulness was found to

have extended nearly to the base of the left lung in front. The softening at the right apex was advancing rapidly. The temperature rose at 10 p.m. to 100·4°, or six degrees Fahr. above the morning temperature. After this she rapidly grew worse, and the ergot was discontinued. Her temperature only once fell below 98°, and in the evening varied from 99·6° to 100°. On the evening of the 21st it reached to 101·4°. The albumen had increased on this day to $\frac{1}{3}$ col., and the sugar was 50 grs. per oz.; the water, however, had fallen to 50 oz. On the 28th she was seized with a severe pain below the left nipple and great dyspnœa; on deep inspiration a friction sound was heard. She gradually became comatose, and died the next morning.

The condensed notes of the post-mortem examination made by my clinical clerk (Mr. E. A. Elkington, M.B.) were as follows:—The rigor mortis was well marked, muscles were a little pale, but free from any fatty change. On opening the abdomen the liver was seen to greatly exceed its normal limits, especially towards the left side. The liver was universally congested especially the hepatic system, the capsule was rather opaque and adherent in some places. The gall-bladder was small and contained only a little brownish bile. The liver weighed sixty-eight ounces. *The peritoneum covering the intestines and forming the omen-*

tum was seen to be minutely injected, the small capillaries being filled with red blood. There were no adhesions of the peritoneum, and no fluid in the cavity. The spleen was healthy in appearance and weighed six ounces; the kidneys weighed nine ounces each. The capsule was easily separable from each, leaving the organ lobulated, with the cortical substance granular to the touch and like dried orange-peel. Over the surface of these organs were numerous branching tortuous veins, the pyramids appeared healthy, the cortex was hard, tough, and granular. The bladder was much hypertrophied, and capable when empty of holding two oranges. The uterus was small, weighing with the ovaries one and a half ounces.

The left pleural cavity contained fully a pint of semi-opaque serum with flakes of lymph; there were numerous recent adhesions; the left lung was smeared over with recent lymph, which glued it to the pericardium on one side, and to the diaphragm below. The left lung when placed in water floated with the base uppermost, its apex was riddled with cavities mostly about the size of a marble; three-fourths of the lung at least was infiltrated with grey tubercular (?) matter, which in many spots had softened and was converted into small purulent depôts. The right lung was in a similar state as regards its apex. There were deposits of yellow cheesy, grey tuber-

cular, and calcareous matter all through it, and one good sized vomica at the base. There was a little bloody serum in the pericardium; the heart was healthy.

The fluid in the left pleural cavity was highly albuminous, *and contained two and a half grains of sugar an ounce.*

A decoction of half an ounce of the liver substance in water showed only a trace of sugar.

Case VIII.—*Treated with diet, and Lactic Acid.*

George W—, æt. 52, a tall, thin man, with red hair, pale complexion, and anxious expression, applied at the hospital suffering from diabetes. His family history was good, and he himself had always enjoyed fairly good health till some twelve months since, when he began to feel greatly fatigued at the end of his day's work, and to be troubled with great thirst. He soon began to lose flesh, and said he had lost 28 lbs. during the last six months. His appetite was good. The tongue was red and fissured, the mouth clammy, the breath sweet, the teeth were carious, the gums tender. He complained of a sinking at the stomach. The liver was not enlarged. The lungs and heart were free from disease. The pulse was 66; the skin dry and rough; the temperature 97°. The urine was copious, and contained a large quantity of sugar, but no albumen.

He was placed on ordinary diet at first, and the following was the progress of the case during his stay in the hospital: the amount of sugar was not estimated:

Ounces of urine in 24 hours.	Sp. gr.	Remarks, Diet, Treatment.
192	1039	Average of 3 days, ordinary diet; no drugs.
90	1035	Average of 7 days, restricted diet; no drugs.
71	1033	Average of 7 days, restricted diet; ʒij lactic acid daily.
103	1035	Last 4 days in hospital, restricted diet; no lactic acid; no drugs.

Case IX.—*Treated with diet, and Lactic Acid.*

John W—, æt. 31, ironcaster, married, reddish hair, fresh complexion. The family history was good. He had always enjoyed good health up to the present illness, and never had rheumatism in any form. He began to feel low and weak some four months before he came to the hospital. He felt unfit for work, had frequently to pass water, and was very thirsty; his appetite was very good all through the illness and has increased of late. He nevertheless had lost flesh to the extent of nearly 28 lbs., weighing now only 103 lbs. The tongue was red and fissured, the mouth parched. The gums were spongy; the teeth, with one exception, sound. The bowels were confined. The liver was not perceptibly

enlarged. The lung percussion was good everywhere, and there were no abnormal sounds on auscultation. Respirations 18 per minute; pulse 72. The heart sounds were healthy. Temperature of body 96·4°. The urine was copious, free from albumen, containing chlorides and a large quantity of sugar. The skin was dry and branny. The progress of the case was as follows:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
180	1047	49	8820	Average of 7 days, ordinary mixed diet; no medicine.
116	1042	36	4176	Average of 14 days, restricted diet; no medicine.
About the middle of this period the lung complications began to manifest themselves at right apex, as pneumonia.				
117	1041	27·5	3217·5	Average of 7 days, restricted diet; lactic acid 45 to 90 minims daily; acid taken irregularly on account of first attack of rheumatism; skin moist and perspiring.
123	1041	36	4428	Average of 14 days, diet as before. Recovering from rheumatic attack. No lactic acid; no drugs. Softening at right apex; free rusty expectoration.
119	1034	26·6	3165·4	Average of 6 days, diet as before; lactic acid 75 minims daily. The acid had to be stopped at end of 6 days, on account of rheumatism.
109	1038	24	2616	Average of 8 days, diet as before; lactic acid taken occasionally, about 50 minims a day. Two slight rheumatic attacks; skin perspiring freely.
Five weeks interval, during which lactic acid was taken irregularly, at the patient's discretion, on account of rheumatism.				

103·6	1041	19·8	2051·3	June 1 to 15, daily average; diet as before; lactic acid 50 minims regularly; lung complications better; slight rheumatic attacks; skin perspiring freely.
97·4	1042	17	1655·8	June 16 to 30, daily average; diet as before; lactic acid 75 minims daily. Skin acting freely. Lung conditions improving much.
110	1041	28	3080	July 1 to 8, daily average; white bread 8 oz.; 75 minims of lactic acid, 5 days; 100 minims 3 days; and stopped on evening of 8th, on account of rheumatic attack. Lung complication stationary.
107	1043	31	3317	July 10 to 17, white bread as before. Sharp rheumatic attack. 100 minims of lactic acid on 10th only. Skin perspiring freely.
98	1041	19·2	1881·6	July 17 to Aug. 2, no white bread, restricted diet; lactic acid 100 to 125 minims daily. Skin acting freely; no rheumatism; lung complications quiescent; harsh breathing at right apex; no expectoration.

Some two months after he left the hospital the lung complications set in again, and he died in three weeks. The lung affection while he was in the hospital was pneumonic in its character, and the expectoration was rusty; with the advent of the lung disease the temperature rose and continued high till the last few weeks of his stay in the hospital, when it frequently fell to 97° in the morning, and in the evening was seldom over 98·4°. During the attacks of joint inflammation the febrile elevation was greater, as I have elsewhere stated. (*The Synthesis of Acute Rheumatism*, page 143.) During the time this man remained

under treatment he gained weight in spite of the febrile attacks; in the middle of June he was six pounds heavier than at the time of admission (February 16th), and 9 lbs. heavier than he was early in March. He lost weight when he took to mixed diet again in July, but regained a little during the last seventeen days he remained under treatment.

Case X.—*Treated with diet, Perchloride of Iron and Bromide of Potassium, and Lactic Acid.*

Mr. John T—, æt. 37, gunmaker; married, with two children; sandy hair; rather sallow complexion; slightly made. The family history was good. He was always healthy till recently, and never had rheumatism nor any other illness except summer diarrhœa. His present illness began some eight months ago when he was in a state of great mental distress at the loss of one of his children. He was troubled with great thirst and passed water too freely. He was tired and unfit to attend to his work, was easily fatigued by walking, and lost flesh although his appetite was very good. His disease was recognised by his family doctor, and he was placed under treatment by diet, with the effect of diminishing the amount of water without improving his strength or general health. When he consulted me he complained mainly of great weakness and in-

ability to attend to his business, increasing emaciation, inordinate appetite, a sensation of heaviness after meals, and excessive diuresis (150 to 200 oz). His expression was anxious, his tongue red, and the mouth clammy; the gums were softish, the teeth sound. The bowels were confined. The thoracic organs were free from disease. The liver and spleen were normal in size. The skin was dry and harsh. Pulse 78; respirations 18. The report of the urine was as follows:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
160	1040	21	3360	Average of 3 days, on ordinary mixed diet; Tr. Ferri Perch. m.x, t. d. s.
79	1034	18	1422	Average of 7 days, restricted diet, *i. e.* meat, fish, eggs, bran bread, water-cress, milk 1 pint, claret 1 pint; Pot. Brom. gr. xx, Tr. Ferri Perch. m.x, t. d. s., Ol. Morrhuæ ʒss bis die.
70	1038	22	1540	Average of 4 days, restricted diet as before, + 2 pints of milk and half a pint of claret; lactic acid 50 minims a day; skin moist.
80	1038	14·8	1184	Interval of 2 weeks, during which the lactic acid was taken irregularly on account of rheumatic pains. The figures are average of 3 days in middle of this period.
74	1038	14·5	1073	Average of 1 week, restricted diet as above; 75 minims of lactic daily; skin moist and perspiring; acid stopped on account of rheumatic attack.
68	1036	12·5	850	Average of 14 days, 18 days interval having elapsed since last series of observations; restricted diet, gluten bread, skim-milk; ʒiss of lactic acid daily for 9 days, ʒij daily for 5 days; no cod-liver oil; no claret.

After this time I did not see Mr. T— for some six weeks, during which period, as I afterwards learned, he followed out the lactic acid treatment at intervals, but never took more than 120 minims in the course of twenty-four hours. He considered that he was improving; and, as I had advised, spent much of his time in the open air, going daily in an omnibus to the outside of the town, where he took a short walk into the country, and then returned home by the same conveyance. On the day but one before he sent for me, he had walked farther than usual into the country, had missed his omnibus, and had walked home. He arrived in a state of great exhaustion. The next day he felt extremely weak and did not go out; he took some purgative medicine which did not act. I saw him on the third day and found him in bed very ill, breathing at the rate of 45 a minute with a pulse of 110. The skin was cold and dry. The lungs were clear all over, and were fully filled by each inspiration. There were no abnormal sounds except loud breathing everywhere, but still he seemed to be breathless; the inspirations were full and deep, but evidently ineffective. This peculiar breathing went on for twelve hours, when he gradually became drowsy and comatose, and died eighteen hours after I saw him; during this period he had passed hardly any water.

Some time after, I witnessed a similar ending to a case of diabetes in a patient whom I saw in consultation with Mr. J. Jackson of this town. A young lady, æt. 29, who had suffered, for some eighteen months, from diabetes brought on by mental anxiety, arranged to visit Birmingham in order to spend the Christmas with her friends. She felt quite as well as usual on the morning of her journey, and equal, she thought, to the effort. She had some distance to drive to the railway station, and then about twenty miles to travel. The train was late, and she was fatigued by the extra time spent in reaching her friend's house. Mr. Jackson saw her that evening, and the next morning I visited the patient, whom I found in bed, very much exhausted and greatly distressed by a feeling of breathlessness. Long, deep, inspirations, which fully inflated the lungs (for they were free from disease) succeeded one another rapidly, but gave no relief. Her intellect was clear, and there was no blueness of the lips. In spite of stimulant remedies, and peroxide of hydrogen, she steadily grew worse and died some twelve hours after I first saw her. She passed urine, in small quantities for a diabetic, during the day, and I found that it contained 25·7 grs. of sugar in the ounce. In both these cases the great diminution in the quantity of urine favoured the accumulation of excrementi-

tious products in the blood, and in this way, I believe, produced a condition analogous to uræmia.

Case XI.—*Treated with diet, Lactic Acid, and Codeia.*

Samuel J—, æt. 39, a married man, thin, slightly built, with light hair, a rather fresh complexion, and an anxious expression. He had served as a soldier, but was discharged on account of shortness of breath. Since then he had worked as a lapidary and enjoyed fair health, except for colds on the chest, till some twelve months ago, when he began to feel stiff about the joints and very tired after his work. He noticed about the same time that he was always hungry and very often felt a sinking sensation at the stomach. His thirst was also great and he passed water very freely. He knew no cause for the illness coming on, except possibly grief at the loss of one of his children. On examination his chest was found to be rather barrel-shaped; it was free from dulness, but slight bronchitic râles were heard both in front and behind. His cough was not very troublesome. His tongue was furred, the gums were spongy, and the teeth carious. The liver was rather large, coming nearly one inch below the costal arch. The spleen was natural in size, the skin dry and branny. There was no œdema of the legs. The urine was free from albumen, but contained a

quantity of sugar. The sp. gr. was 1045. He was first treated as an out-patient and was afterwards admitted into the hospital. While an out-patient he lost 6 lbs. in weight, in one month. It was found impossible to keep this man on thoroughly restricted diet; he helped himself constantly to the bread of other patients when it was attempted. The progress of the case was as follows:

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
172	1039	28·5	4902	Average of 4 days, diet partially restricted, *i.e.*, skim milk 4 to 5 pints, meat, 1 egg, bread 6 oz.; no medicine, except Ol. Morrhuæ ʒij t. d. Temperature 97·5.
117	1038	26·8	3135·6	Average of 7 days, diet as above, except skim milk 3 pints; lactic acid ʒij daily. Temperature 97·5 to 98·2.
100·5	1040	23·8	2391·9	Average of 7 days, diet as above, except skim milk 3 pints; lactic acid ʒj to ʒiss daily. Skin moist. Temperature normal.
96	1039	26·5	2544	Average of 6 days, diet as above, except skim milk 3 pints; lactic acid ʒij to ʒiij daily. Skin perspiring freely. On 6th day the acid was stopped on account of purging.
117	1040	27·6	3229·2	Three days interval; average of 5 days, diet as above; no lactic acid. Gained 2¼ lbs. during whole period of treatment.

This man remained in the hospital some ten days longer, and during the last eight he took codeia, at first in doses of a quarter of a grain

twice a day, and after the third day to the extent of 4 grs. daily; more he could not take on account of the vertigo and unpleasant head symptoms which came on. The diet was unchanged. When he began the codeia he was passing 120 oz. of urine; sp. gr. 1038, and containing 28·4 grs. of sugar in the ounce: on the eighth day the urine had fallen to 84 oz., sp. gr. 1032, with 20·5 grs. of sugar in the ounce. In other words, the total amount of sugar excreted daily had fallen during the week from 3408 grs. to 1722 grs. The diminution was steady from day to day, and coincided with an increase of weight of nearly 3 lbs. The codeia pills were continued for some time after he left the hospital, and the man improved still more, gaining weight and being able partly to resume his work. Three months after his discharge his weight was 124 lbs., or 7 lbs. more than when he entered the hospital.

In estimating the effect of the lactic acid on the quantity of urinary water in this case, it must be remembered that a large amount of fluid was taken as medicine:—no less than 48 oz. in the twenty-four hours, during the period of the largest doses of lactic acid. The acid was dissolved in that quantity of water, and 2 to 4 oz. of the mixture taken every hour. In spite of this, the excretion of urine fell, but the functions of the skin became more active from day to day,

and in this way the decrease in the quantity of urine and sugar is most probably explained.

The lactic acid was given in the quantities mentioned, in order to test the influence of large doses on the urinary excretion, and further, to determine, if large doses would develop rheumatic symptoms after small ones had failed to do so. The results show that very large quantities of the drug may be given without the development of rheumatism. In this last respect the results confirm those obtained by Dr. George W. Balfour, who has published some valuable remarks on the use of lactic acid in diabetes.[3] In several instances in which lactic acid has been given to my out-patients there has been no occurrence of rheumatic pains. The case of Wright (Case IX), however, was such a striking example of the manifestation of rheumatic symptoms over and over again under the use of lactic acid,—the inflammation of the joints being perfectly under the control of the medicine, coming on when it was taken and gradually passing away when it was discontinued,—that I can have no manner of doubt as to the influence of the acid in developing rheumatic fever under favourable conditions. It is to the exact definition of these favourable conditions that future inquiry must be directed in order to settle the pathology of acute rheumatism.

(3) *Edinburgh Medical Journal.* Dec., 1871.

CLASS III.—DIABETES FROM ABNORMAL GLYCOGENESIS.

Patients suffering from this form of diabetes are generally weak, thin persons, with an anxious, depressed-look, who have no previous history of a period of increased nutrition. They often complain of a feeling of chilliness, and have always a lowered state of bodily temperature, even when the quantity of urine is not great, and the disease is comparatively recent. The weakness and emaciation of the patient are out of proportion to the duration of the malady and to the quantity of sugar excreted. It seems that the chief characteristic of the condition of these patients is a profound alteration of nutrition, associated from the beginning with a greatly increased excretion of urea. In the treatment of this form of diabetes, any drug which checks the increased excretion of urea greatly benefits the patients. In this way opium, arsenic, bromide of potassium, and particularly valerian, are valuable. The most rational method of treatment, however, yet proposed is that which rests the liver by a purely animal diet, and at the same time administers some easily combustible substance, such as lactic acid or glycerine, to replace the faulty liver sugar, and save, by its combustion, the albuminates and fats. Skim milk

diet also does this, and hence its value. Of the cases that follow Nos. 12 and 13 are examples of the fully developed disease, and 14, 15, and 16 are specimens of its earlier stages.

Case XII.—*Treated with diet, Opium in small doses, Ether, Glycerine, and Permanganate of Potash.*

T. M., æt. 16, brassworker. The patient comes of a healthy family, and never had any serious illness till the present one, which began about twelve months before his admission to the hospital. The symptom which first attracted his attention was weakness during his work, for which he could not account; about the same time his appetite failed him, he began to lose flesh, and was very thirsty. For some months he did not notice that his water was in excess. On admission, he was about five feet one inch high, with light sandy hair and fair complexion, a worn and anxious expression, and weighed only 62 lbs. He was very greatly emaciated; his skin was dry and branny; his fingers were clubbed; his sight was dim, and he complained of attacks of giddiness, but not of headache. He had never suffered from cough, and never had swelling of his feet. On examination, the lungs and heart were found to be healthy. His abdomen was a little distended, the superficial veins being very distinct all over its surface. The liver appeared to be smaller than natural, and ex-

tended only as low as the tenth rib. The spleen was natural. There was no tenderness over any part of the abdomen, nor over the kidneys posteriorly. The tongue was moist, red, and fissured; the appetite was large. The bowels were confined. The urine on admission was loaded with sugar, and had a sp. gr. 1042; he passed about eight pints a day. The progress of the case was as follows:—

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
160	1040	48	7680	Average of 4 days ordinary diet.
138	1036	43	5934	Average of 7 days restricted diet, composed of meat, cabbage, 4 pints skim milk, bran bread (18 oz.), 1 pint of beef tea. Treatment, hot bath twice a week. 1 gr. opium twice daily.
104	1032	40	4160	Average of 7 days same diet; 1 gr. of opium daily; gain 1½lbs. in weight.
96	1034	38	3648	Average of 14 days same diet; bran biscuit instead of bran bread occasionally. Treatment 20 m. of spt. of ether three times a day. Cod liver oil a drachm twice a day.
116	1031	28·25	3277	Average of 7 days, diet less restricted; more milk and bran bread. Ether continued. 2 lbs. more in weight gained at the end of this period.
125·25	1027	26·6	3331·6	Average of 10 days, same diet. Glycerine 1 to 2 oz. daily in water. Liq. pot. permang. ʒi in water three times a day.
104	1030	27·5	2860	Interval of 4 days. Average of 14 days, same diet. Glycerine alone given; permanganate discontinued. Weight at the end of this period 68¼ lbs. Total gain, 6¼ lbs.

After this time pulmonary complications set in. He had pleuritic attacks as well as Diabetic Phthisis. His temperature during the most active lung mischief never reached 100° F. As the lung disease advanced his urine diminished in quantity, and towards the last often fell as low as 60 oz., but seldom contained less than 40 grs. of sugar in the ounce. On two occasions, when it fell to 57 and 55 oz., *there was a copious deposit of urates*, the quantity of urine being no longer sufficient to hold them in solution. This unusual appearance shows that we cannot, as Prout thought, always refer the commencement of diabetes to the last appearance of a deposit of urates in the urine.

Case XIII.—*Treated with diet, and Peroxide of Hydrogen.*

John S—, æt. 29, iron-roller, married and the father of three children; a tall man, with sandy hair and a rather florid complexion. The family history was good. The patient was always healthy till about two months before admission, when he felt weak and had pains in his limbs after work. He noticed that his thirst was great, and his water increased in quantity. He considers that his illness was brought on by worry over family troubles. He passed at one time as much as six pints of water in the night. He had lost

twenty-one pounds in weight since he began to be ill. On examination his lungs and heart were found to be healthy. The tongue was red at the tip and the edges, white at the base, and rather dry. The gums were rather spongy, the teeth sound. The skin was dry and harsh. He complained of pain in the frontal region of the head, which he says has troubled him all through his illness. His weight was 124 lbs. The temperature of the body varied from 96·4° to 97·8°, the highest point observed during the whole of his stay in the hospital; once it fell to 94·4°. The progress of the case was as follows:—

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
217	1037	36·8	7985·6	Average of 7 days on ordinary mixed diet.
128	1040	42	5376	Average of 7 days, partially restricted diet, *i. e.*, bran bread, green vegetables, meat, milk; ʒss Tr. Ferri Perch., daily.
101	1038	38	3838	Average of 7 days, diet as before, skin softer; peroxide of hydrogen, ʒij daily.
84	1028	23	1932	Average of 9 days, restricted diet, *i. e.*, no bran bread; ʒiij of peroxide of hydrogen daily; copious perspirations.

This patient remained some weeks longer in the hospital, but as he could not be kept wholly on starchless food he was allowed Blatchley's bran-cakes. At the time of his discharge he was pass-

ing 75 oz. of water (on an average) containing 22 grs. of sugar per oz. He continued the peroxide of hydrogen, which he said did him more good than anything. While under the influence of this drug he perspired copiously, and the perspiration contained sugar in considerable quantity—on one occasion as much as 8 grs. per oz. The perspirations were so free that the secretion could be easily collected in a test tube. He gained while under treatment 3 lbs. in weight.

Case XIV.—*Treated with diet, Perchloride of Iron and Bromide of Potassium, and skim milk.*

Mr. W. R. W—, æt. 30, merchant's factor, a tall slight man, of a dark complexion. He was born in Australia, whence he came to England three and a half years ago. He always enjoyed good, though not strong health, till about three months ago, when he received a severe blow over the last two dorsal vertebræ. He did not take much notice of the injury at the time, but has frequently had dull pain there since. The illness began with a feeling of languor, considerable thirst, and a large secretion of urine. The appetite was large, but in spite of this there was a steady loss of flesh; the bowels were inactive.

On examination no trace of injury could be found on the back; there was tenderness on pressure over the left kidney; the lungs and heart

were healthy; the liver and spleen were normal in size; the tongue was red, moist, and fissured; the gums were spongy; the right molar teeth carious; the skin was dry and hard. The temperature was 97·5° F. The urine has been as much as five pints daily, but is now less. The progress of the case was as follows :—

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
76	1038	30	2280	Average of 3 days on partially restricted diet.
64	1035	24·5	1568	Average of 1 week, diet partially restricted; taking Tr. Ferri Perchlor. ʒss and bromide of potassium 40 grs., daily.
42	1040	15	630	Average of 1 week; same medicine; strict diet.
40	1030	3	120	Average of 1 week; same medicine; skin moist and perspiring; strict diet.
38	1029	Trace		Two days average, strict diet, same medicine. Between this time and the previous observation, an attempt to take some white bread on one day, as part of diet had caused the sugar to rise to 12 grs. per oz., the water to 57 oz.
Four months later Mr. W— came back to me with all his old symptoms. A return to ordinary mixed diet had caused the relapse. He passed at this time—				
72	1037	21	1512	3 days average on mixed diet.
47	1035	18	846	1st week, daily average, skim-milk treatment.
56	1029	7·5	420	2nd week, daily average, skim-milk.

After this he improved still more, but I had no opportunity of again testing the urine. Later on

he relapsed, through a return to mixed diet, and I am informed that he died in about twelve months after the last record of the state of the urine.

Case XV.—*Treated with diet, Lactic Acid, and skim milk.*

Mr. T. W—, æt. 45, single, artist, was sent to me by my colleague Mr. Oliver Pemberton. Mr. T. had always lived temperately and enjoyed good health up to about two years before he consulted me. He referred his diabetic symptoms to mental distress caused by his mother's death. The illness began with weariness, indigestion, thirst, and frequent micturition with increase in urinary secretion. His appetite continued good, but he steadily lost flesh, being when first seen about 22 lbs. below his ordinary weight. For eighteen months past he had lived on partially restricted diet consisting of meat, green vegetables, 6 oz. of toasted bread, and claret. During the last month he had lost nearly 2 lbs. in weight. He complained of feeling chilly, and on investigation his temperature was found to be only 97·4° F.; on several occasions it was found to be under 98° F., and twice was observed, on a warm day, to be as low as 96·5° and 96·8°. The tongue was furred, with red edges; the gums were spongy; the teeth decayed. The appetite had not been quite so good lately. The heart and lungs were free from disease; the liver was

not perceptibly enlarged. The skin was dry and rough. He weighed 122 lbs. The following table shows the state of urine when he was first seen, and at the end of four weeks after he had been placed on restricted diet and lactic acid. The figures in each line, except the first, represent the weekly average. During the month he gained 3 lbs. in weight. He continued to improve after this date, but I had no opportunity of estimating the quantity of sugar in the urine. When last I heard of him, some two and a-half years later, he was enjoying comparatively good health:—

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
130	1033	25	3250	Three days average, partially restricted diet; no treatment.
120	1032	24	2880	Average for 1st week, restricted diet. 2 pints of skim milk; lactic acid ℥j daily.
112	...	19	2128	Average for second week, ditto.
90	1032	15	1350	Average for 3rd week, diet the same; ℥ij of lactic acid in 24 hours.
84	1030	12	1008	Fourth week, ditto.

Case XVI.—*Treated with diet, skim milk, Lactic Acid and Opium, Arsenic, and Valerian.*

T. G—, æt. 50, married, labourer, a large-boned, muscular man, with a rather florid complexion and anxious expression, came to the hospital complaining of great thirst, frequent micturition, and pro-

gressive weakness and wasting in spite of a large appetite. He had never suffered from any serious illness before, and referred his present attack to mental anxiety and trouble connected with his work. The first symptom he noticed was weakness in his limbs, often associated with a burning sensation in them. This was followed by excessive thirst and frequent micturition; this latter symptom compelled him to get up five or six times in the night. His mouth was parched, his skin was dry and harsh, and his bowels were confined. He considers that he has lost many pounds during the three months which have elapsed since he was taken ill. When admitted his heart and lungs were free from disease. His tongue was dry, furred at the back, but red at the tip and edges; his gums were spongy. The sight was good. He did not suffer from headache. The liver and the spleen were natural in size. There was no tenderness over the abdomen or in the lumbar regions posteriorly. His temperature was 97·4. His weight was 129½ lbs. The urine was large in quantity, contained 30 grs. of sugar to the ounce, and had a sp. gr. of 1,042. He was placed on ordinary mixed diet. The progress of the case under the various plans of treatment adopted is tabulated below:—

Ounces of urine in 24 hours.	Sp. gr.	Grains of sugar per oz.	Daily amount of sugar in grains.	Remarks, Diet, Treatment.
148	1043	33·4	4943·2	Average of 4 days on ordinary mixed diet. Inf. gent. ʒi t d s.
119	1038	23·5	2796·5	Average of 7 days on restricted diet of meat, skim milk, beef tea, bran bread, and green vegetables. Inf. gent. ʒi t d s.
99	1033	18	1782	Average of 10 days on skim milk and curds. Inf. gent. as before. On the last day of this treatment the sugar fell to 13 grs. per oz. The temperature during the 10 days rose to and kept at normal line. The man refused to continue the skim milk beyond the 10 days.
124	1040	25·5	3162	Average of 14 days on diet of meat, bran bread, green vegetables, and 3 pints of skim milk. Treatment, lactic acid ʒi daily, and 3 to 5 grs. of opium a day. Left hospital at end of a fortnight, weighing 132 lbs.
150	1037	27	4050	Average of 7 days; living at home on same diet as last 14 days of stay in hospital. Treatment: Liq. arsenicalis, 10 to 15 minims daily.
110	1035	27·5	3025	Average of 11 days, diet of meat, cabbage, milk, eggs, bacon, bran bread. Treatment: Inf. valerian 6 oz., and 50 grs. of ext. of valerian daily.
116	1034	26	3016	Average of 3 weeks, same diet. Treatment: Infus. valer. 5 to 6 oz., and 80 to 90 grs. of ext. valerian daily. Weight, 147½ lbs.

The table gives a by no means sufficiently favourable view of this patient's improvement after he left the hospital. It shows how much he improved on the skim-milk diet, which reduced the amount of sugar on one day to as little as 13

grs. per oz. He absolutely refused, however, to continue this diet, and left the hospital when I again pressed it. During the last fortnight of his stay the so-called galvanism of the sympathetic nerve in the neck was tried, but produced no appreciable effect on the flow of urine or the sugar excretion. When he left the hospital, he had gained about $2\frac{1}{2}$ lbs. during his stay. The first week at home, where he had a freer diet, considerably increased the amount of sugar, and the arsenic appeared to have no power of checking the excretion; the arsenic was continued for a longer time than appears in the table, having been given altogether for 14 days, but no analysis was made during the first week. He was afterwards placed on the valerian treatment, and on this he gained no less than $15\frac{1}{2}$ lbs. in weight. The thirst decreased. His strength increased; he was able to do a fair day's work; he felt better and stronger, and improved in appearance, although the diet which he used at home contained more starch than the restricted diet of the hospital. On two or three occasions he was ordered to take liver as part of his animal food. This I did, because it seemed to me that the liver offered a ready way of introducing healthy liver-sugar to supply the deficiency of combustible material which exists in cases of diabetes from abnormal glycogenesis. While taking the liver there was no increase in the quantity of

urine nor in the amount of sugar. On two occasions on the days following a free use of liver the urine was 115 and 118 oz., with 25 and 23 grs. of sugar in the ounce. During the period of treatment as an out-patient his water was examined three times a week, on Tuesdays, Fridays, and Saturdays. The results recorded in the table show that under the use of the valerian, the improvement in general health, and the increase in weight, were out of proportion to the diminution of the sugar. I was disappointed time after time to find the quantity of sugar so large while the man was manifestly improving. To see how far this might be due to the effect of the valerian in checking the loss of urea, I made four analyses of the urine during the last three weeks the patient was under observation, and the comparison of the results with an estimate of the urea made shortly after he left the hospital, explained the mystery. When the first analysis was made, before the valerian treatment was commenced, he was passing daily 907·5 grains of urea.

During the last three weeks of the valerian treatment the amounts found were—	732·4	„	„
	678·	„	„
	640·3	„	„
	596·8	„	„

These figures, although far from conclusive, show, I think, that valerian, in the first place, produces its beneficial effects rather by checking tissue waste than by lessening the sugar excretion.

IV.

In addition to their interest as illustrations of the effects of remedies on the glycosuria, the foregoing cases possess other noteworthy features. Of these I may refer to the frequency of mental anxiety and grief as antecedent conditions. No less than eight of the sixteen patients whose cases are recorded, referred their illness to this cause, and the lady to whom reference is incidentally made under Case X, was another example of the malady supervening on mental trouble. Prout considered mental anxiety or distress one of the most frequent exciting causes, and my experience leads me to regard it as the most frequent of the commonly mentioned antecedents of diabetes. One of the cases (XIV) is an illustration of the disease being excited by bodily injury. There was no reason whatever to suppose that this gentleman suffered from any ailment before the accident to which he attributed his diabetes. Similar injuries to the dorsal region have been known to be followed by permanent glycosuria, and we are justified in regarding this case as an example of the disease resulting from a somewhat rare cause.

The state of the temperature of the body noted in these cases is, however, the phenomenon of the greatest interest. It is an extremely curious

clinical fact that patients, excreting urea abundantly and wasting rapidly, should present in their low temperature a contrast to the usual results of tissue destruction. Here we have a process of tissue wasting equal to that seen in fevers, but nevertheless associated with a depression of the bodily temperature, which is often lowest when the wasting is greatest and the urea excretion highest. Some observers have been so impressed by the excessive urea-loss (azoturia), that they have elevated it to the position of the prime evil of diabetes, and relegated the glycosuria to a secondary place. According to this view, the disease really consists of a profound disorder in the nutrition of the albuminous textures which permits their constant disintegration and produces the azoturia. To this azoturia the glycosuria is added, and together they constitute a form of diabetes which is always grave by reason of the rapid wasting. The diabetes, on the other hand, in which the excessive excretion of urea is not present, is less serious in its character. This close connection of azoturia with diabetes was long ago detected by the sagacity of Prout, who considered that an excessive loss of urea might precede the appearance of sugar in the urine. Without accepting the extreme views, which elevate the urea-loss to the first rank in diabetes, we have much evidence in recent phy-

siological work to show that urea, like sugar, is mainly a product of liver cells, and may consequently, it is highly probable, be formed in excess under circumstances which augment the activity of the liver circulation. A great part of the urea excreted in cases of excessive glycogenesis may, however, be accounted for by the animal diet taken; the peptones derived from the albumen of this diet being split up into urea and glycogen, both of which are excreted. Later on, a disintegration of the albuminous tissues themselves occurs, and the azoturia becomes a leading feature of the malady, as it does from the beginning in cases of abnormal glycogenesis. It is a fact of much interest that in diabetes insipidus there is often a large increase in the amount of urea excreted, which is strangely enough associated with a lowered bodily temperature. In a case of diabetes insipidus, in a youth aged eighteen years, I have observed an average daily temperature of 97·2° to 97·8° coinciding with a daily excretion of 664·4 to 642·7 grs. of urea, when the normal amount of urea, considering the boy's weight, ought not to have exceeded 320 grs. a day.

Apart, however, from the curious association of low temperature with excessive urea-excretion in diabetes mellitus, the relation of the temperature in this disease to other conditions and its special peculiarities are matters of much interest.

Some time ago I called attention to this subject in a "Note on the Temperature in Diabetes" in the *Journal of Anatomy and Physiology*.[4] In that note I stated the conclusions at which I had then arrived. I now reproduce these conclusions, with the modifications and additions which a more extended study warrants.

The temperature in diabetes had, I believe, been previously studied by Rosenstein, but as I have not seen his papers, I am unaware of the nature of his work. The conclusions which are advanced above are based entirely on my own observations, which were begun in 1864, when my attention was first directed to the subject by the absence of any decided febrile elevation in a case of well-marked diabetic phthisis.

I.—The temperature is always below the normal standard in advanced cases of diabetes in which there are no inflammatory complications. The depression of temperature varies from 1° to 5° F. In the class of cases which I have called defective glycocrypsis, the temperature is generally normal, but may fall a little when the quantity of urine is large. In the early stages of cases of excessive glycogenesis, while there is increased nutrition or no wasting, the temperature is generally maintained at the healthy standard, though occasionally it may be depressed.

(4) Vol. iii, part 2, May, 1869.

It is in the second stage, or when wasting has set in, that we have the fall in temperature most decided. It then equals the depression of temperature observed in cases of abnormal glycogenesis, and may fall as low as 94° F. or even lower.

In recent cases of abnormal glycogenesis, the temperature is often only 1° to 2° below the normal standard, but as the case advances the depression usually increases, and it is in this form of the disease that some of the lowest temperatures are seen.

II.—The evening temperature is often, but not invariably, higher than the temperature in the morning by one half to eight-tenths of a degree. This is more especially seen in the earlier stages, before the wasting has set in. In advanced cases the temperature has been frequently observed to fall in the evening.

III.—The temperature is not proportional to the variation in the quantity of sugar. The same temperature has been observed over and over again in the same cases to coincide with widely different quantities of sugar.

IV.—The daily range of temperature bears no proportion to the quantity of sugar passed in the twenty-four hours, as seen in the following table:—

Case I.			Case II.		
No. of Observation.	Daily range of temperature.	Quantity of sugar in 24 hours.	No. of Observation.	Daily range of temperature.	Quantity of sugar in 24 hours.
1	$\frac{6}{10}°$	920 grs.	1	$\frac{4}{10}°$	1856 grs.
2	$\frac{8}{10}°$	2310 grs.	2	$1\frac{2}{10}°$	660 grs.
3	$\frac{2}{10}°$	3640 grs.	3	$\frac{6}{10}°$	792 grs.
4	$1\frac{2}{10}°$	2730 grs.	4	$1°$	1320 grs.

In one case observations were made in the following manner:—The bladder was emptied one hour before the thermometer was used, and emptied again about half an hour after the temperature had been recorded. The quantity of sugar per oz. in the urine thus procured was compared with the temperature with the results recorded below:—

Observation.	Temperature.	Sugar per oz.
1	97.4	32 grs.
2	96.8	19 grs.
3	97.2	23·5 grs.
4	96.5	33 grs.
5	96.7	24 grs.

V.—The administration of a mixed diet in cases of excessive glycogenesis is often followed by an elevation in temperature. The enforcement of a strictly animal diet, on the other hand, is frequently observed to coincide with a lowered state of temperature, in which the daily range is less: that is to say, the morning and evening

temperatures are nearer. In one case in which temperature observations were made every half-hour during the day, an increase in the temperature was observed to occur from one to two hours after meals. The patient was fed on mixed diet during the observations.

VI.—The occurrence of inflammatory complications in diabetes produces an elevation of temperature, but to a very limited extent when compared with the temperatures observed in the same inflammatory affections in persons not diabetics. The highest febrile temperatures which I have recorded in diabetics have been 101·2° in rapid phthisis, and 101° in acute rheumatism supervening during the administration of lactic acid. These high temperatures were observed in cases of excessive glycogenesis. In a case of abnormal glycogenesis, with well marked phthisis and local pleurisy, the temperature was seldom higher than 99°, and only once reached 99·4°.

The depressed state of the temperature in diabetes has given rise to many speculations respecting its cause. The large quantity of cold water which the patients drink naturally suggests itself as a possible explanation of some of the loss of body heat. A man who drinks a gallon or more of cold fluid with a temperature say of 50° to 55° in the course of the day, must necessarily consume some of the heat he produces in raising its

temperature to that of his body. This idea has, I believe, been advanced by no less an authority than Bouchardat. In order to test it, I compared the average daily temperature in several cases with the quantity of water excreted in the twenty-four hours. The results which follow show that there is a relation, though not a very constant one, between the quantity of urinary water and the average daily temperature :—

FIRST SERIES OF OBSERVATIONS.

Day.	Average daily temperature (three observations daily).	Ounces of urine in 24 hours.	
1	97·7°	212,	mixed diet.
2	98°	184	partially restricted diet.
3	98·1°	90	
4	98·3°	92	
5	98°	84	
6	98°	92	
7	98·3°	94	
8	98·3°	84	

SECOND SERIES OF OBSERVATIONS.

Day.	Average daily temperature.	Ounces of urine in 24 hours.
1	97·6°	164
2	97·7°	174
3	97·5°	188
4	97·2°	124
5	97·1°	124
6	97·4°	128
7	97·6°	112
8	97·7°	72
9	97.9°	96
10	97·7°	100

In making these observations, I was quite aware

that in diabetes the quantity of urine passed in any one day is a most fallacious guide to the estimation of the quantity of fluid taken, and I therefore extended the observations over a number of successive days. The discrepancy between the quantity of urine passed on a given day and the amount of liquids taken as drink, formerly led observers to hold the opinion that diabetics passed more water than they drank. It was supposed that the surplus was obtained by absorption of watery vapour through the skin and lungs, or by the liquefaction of the tissues of the body. Thanks to Nasse, Griesinger, and others, we now have more precise knowledge on this point. The urine never exceeds the drink in quantity if the comparison extends over a sufficiently long period. On a given day, indeed, a diabetic patient may pass more water than he drinks, and this was the source of the erroneous opinion formerly held. This discrepancy is, however, only temporary, and has been admirably explained by Vogel. It seems that in diabetes the fluids taken are passed out of the body more slowly than in health. This is due to the fact that whenever a diabetic is very thirsty, his blood has been becoming for some time more and more concentrated in consequence of the continual flow of water outwards through the kidneys. The sp. gr. of the blood also rises on account of the continued entry of sugar into it, and therefore

draws water from all the tissues outside the blood vessels, till the fluid, bathing the tissues, becomes as dense as the blood itself. When drink is taken the blood is diluted by its absorption, and then the tissues in their turn receive a share of the water, till the fluid in their meshes is reduced to the sp. gr. of the blood. As the urinary secretion, however, constantly goes on, the blood again becomes more dense by its loss of water, and again absorbs the fluid stored up in the tissues. All this is a contrast to the healthy state in which the increased vascular pressure produced by the ingestion of an extra supply of fluid is almost directly relieved by the increased flow of urine.

In order to test still further the influence of cold drinks on the temperature, I persuaded one of my patients to take all his fluids warm for six days. The following table shows the results :—

THIRD SERIES OF OBSERVATIONS.

Day.	Average daily temperature.	Ounces of urine in 24 hours.	Remarks.
1	97·3°	100	Two days cold drinks.
2	97·1°	92	
3	97·7°	112	Six days all fluids, warm.
4	98·4°	100	
5	98·3°	112	
6	98·3°	112	
7	97·9°	96	
8	98·4°	100	
9	98 2°	100	All fluids, cold.
10	97·2°	96	

The results contained in the last table were obtained when the patient was kept on partly restricted diet, and took a drachm and a half of lactic acid daily. The conditions were, as far as possible, perfectly the same during the whole period, and yet the patient showed a decidedly increased temperature throughout the period during which the warm fluids were taken; while the temperature fell again when they were discontinued.

These results are, I think, sufficient to justify the conclusion—

VII.—That the quantity of cold fluid taken has a slight, but decided influence on the body-heat in diabetics.

The cases which I selected for these experiments were examples of excessive glycogenesis, in which the slightly depressed temperature rendered it probable that the second stage of the malady was commencing. The autophagia, although not marked, had begun. These results, I think, explain how it is that in some cases of defective glycocrypsis, when the urinary water is copious and the thirst great, we observe a lowered temperature; they also explain the low temperature observed occasionally in the early stages of cases of excessive glycogenesis, when 200 to 300 oz. of urine are passed daily. Under such circumstances, even when there has been no decided wasting, I have observed a temperature as low as 96·5°.

Soon after I began to study the temperature in diabetes mellitus, I found that in cases of diabetes insipidus there was also a lowered temperature. In this last disease the depression is slighter, and I have never observed it lower than 96·2°. For some time this depressed temperature occurring independently of glycosuria puzzled me very much. The results I have recorded above, however, pointed to the explanation, and I have since found in a case of diabetes insipidus a close relation between the average daily temperature and the quantity of urinary water.

In the advanced stage of diabetes from excessive glycogenesis, and from an early period in cases of abnormal glycogenesis, the temperature is not closely related to the quantity of urinary water. Very low temperatures may be observed to coincide with comparatively small daily amounts of urine. The reason of the low temperature lies indeed much deeper than the quantity of fluid taken, and is to be sought in the altered conditions under which the body heat is maintained. The natural fuel of the body is no longer formed by the liver, and the combustion of the albuminates and fats of the food and tissues but inadequately supplies its place.

IX.

CASES ILLUSTRATING THE USE OF THE SPHYGMOGRAPH AND CARDIOGRAPH IN THE STUDY OF DISEASES OF THE HEART AND GREAT VESSELS.

In this article are collected a few cases which have been studied with the aid of the sphygmograph and the cardiograph. Whatever interest they possess is due to the method of clinical investigation pursued, and to the facts recorded in illustration of the value of these instruments of precision in the study of diseases of the heart and great vessels. By appending to each case an abstract of the clinical remarks which it suggested at the time of its occurrence, I have condensed into a comparatively small space the substance of several clinical lectures.

Case I.—*Aortic valve disease, with dilatation of the aorta—Examination with the sphygmograph and cardiograph—Death—Autopsy—Clinical remarks.*

J. P., æt. 36, married, of Hunnington, a farm labourer, came into hospital complaining of great

difficulty of breathing and frequent epistaxis. He stated that he had been ill since last Christmas, when he had a low fever, which lasted several weeks. Previously he had suffered from occasional rheumatic pains in his limbs, but never from rheumatic fever. Up to the last year or so he had always enjoyed good health, and had lived temperately. Since his illness last Christmas he had been subject to shortness of breath, attacks of pain in the chest, and paroxysms of difficult breathing. About eight weeks back an attack of epistaxis occurred and lasted some fourteen hours, and had since recurred nearly every week. He had not been able to sleep soundly for some time on account of noises in his head and horrible dreams, and whenever he had slept well for a little time an attack of dyspnœa occurred on waking.

Two days after admission the following notes were made: The man sat up in bed, supported by pillows, on account of his dyspnœa. The skin was of a yellowish earthy colour; the expression anxious, the breathing quick and laboured. He spoke in a short gasping manner, and complained of an almost constant sickness, which distressed him very much, of frequent bleeding from his nose, and occasional cough. He had scarcely slept since admission, as whenever he had, so to speak, surprised his disease in a moment of remission and dozed off, he awoke in a few seconds,

with a start, to fight for breath. The tongue was pale and anæmic, and dry from breathing through the mouth alone (the nares having been plugged for the epistaxis); the feet were œdematous; the respirations were 30 a minute; the pulse was 80, jerky, and visible in the arteries. Scarcely any difference was found by the finger in the two radials—if anything, the right seemed the smaller; the temporals were equal to the touch; the pupils were equal. Pulse-traces were taken, and found to be as follows:—

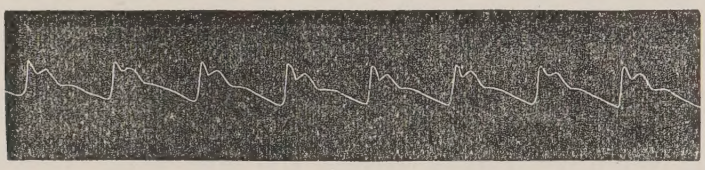


Fig. 1.—Right radial.

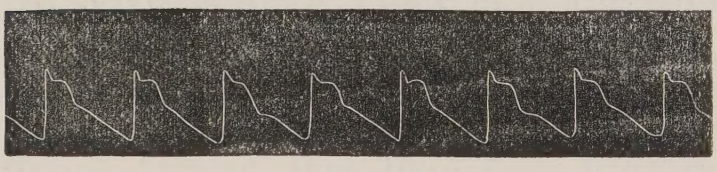


Fig. 2.—Left radial.

On examination the percussion sounds over the lungs were normal; the respiratory murmur was accompanied only by occasional rhonchus; the cardiac dulness was considerably increased, extending from the middle of the third intercostal space on the left side downwards to the lower border of the seventh rib, and laterally from half an inch beyond the left nipple to two inches

beyond the right edge of the sternum; the impulse was heaving and diffused. In the interval between the second and third costal cartilages on the right side was noticed a point of pulsation; this pulsation affected the next intercostal space below, and was felt by the hand to extend to within about one inch of the right nipple. The pulsation immediately followed the heart's systole, and gave an apparent motion to the right side of the thorax from the lower margin of the second to the sixth rib.

On auscultation the heart's sounds were heard as follows: At the apex the first sound was loud and clear, but the second sound was marked by a murmur which nearly filled up the long pause, and increased in intensity as the base was approached. This murmur was more audible over the right ventricle and at the ensiform cartilage. At the base a loud double murmur was heard. The systolic murmur was loud and rough; the diastolic murmur, loud and long, replaced the second sound, and was only terminated at the base by the succeeding first sound. Both murmurs were loudest at the junction of the third right costal cartilage with the sternum. Over the pulmonary artery they were not so distinct, and the pulmonary second sound could be distinguished. The two murmurs could be heard close to the right nipple, along the great vessels under the clavicles, and

at the sternal notch. They could be also heard posteriorly on both sides of the back of the chest. The liver was found to be somewhat enlarged; the urine was high-coloured, scanty, and contained a little albumen and a few casts.

Three days later a careful examination of the chest discovered the same conditions as above described; the heart's action, however, was somewhat weaker. A slight murmur could be detected with the first sound at the apex, and from its nature and position it was referred to secondary mitral insufficiency consequent on dilatation of the ventricle. The urinary secretion was very scanty and slightly albuminous; the œdema of the legs had increased. The patient had had no sleep; had frequently vomited during the night; and was breathing about thirty times a minute. He complained of a noise in his head like a forge at work. There was slight dulness posteriorly at the bases of the lungs, and moist sounds were heard all over the chest. He was ordered a stimulant draught every two hours; dry cupping over kidneys, and afterwards hot poultices. The pulse-traces were taken and found as before. The cardiograph was applied to the apex beat and to the point of pulsation on the right side of the thorax. The traces taken are figured farther on, Nos. 4 and 5.

After this time the patient gradually sank, the

heart's action failing more and more, and the mitral murmur becoming more developed. Total suppression of urine occurred, with frequent vomiting, and the patient fell into a semicomatose (uræmic) condition, and died on August 23.

The post-mortem examination was made thirty hours after death. The legs were œdematous; the body had a peculiar earthy yellow tint, and gave out a strong odour of commencing decomposition. On opening the thorax, the aorta was found large and flaccid. There was some fluid in the pleural cavities. The lungs were œdematous, and congested posteriorly. The liver was large and congested. The kidneys were œdematous; the cortical portion was of normal depth, but tough, granular, and discoloured by commencing decomposition. The spleen and other viscera were healthy. On opening the pericardium, the heart was found to be very large, the left ventricle being greatly hypertrophied. The left cavities contained blood and fibrinous clots. Some clots were also found in the right cavities.

The heart and aorta were carefully examined seven days after the post-mortem examination. The heart was large, weighing eighteen ounces; the increase was chiefly due to hypertrophy and dilatation of the left ventricle. The aorta was much dilated at its origin and in the ascending portion, and formed anteriorly and towards the

right side a dilated pouch. At the termination of the ascending portion, however, the dilatation was no longer marked, and at the origin of the great vessels of the neck, the diameter of the vessel was but little more than normal. On looking down the aorta, the aortic valves could be seen to be deficient, forming a small irregularly shaped aperture through which fluid found easy access to the ventricle. On opening the left ventricle, the following lesions of the valves were found: the right coronary and posterior segments of the aortic valves were united and much diseased. Their edges were rounded and one-eighth of an inch in thickness. This thickening was hard and fibrous in the right coronary segment, and the edge of the posterior segment was studded with calcareous particles on both the aortic and the ventricular surfaces. A small portion of the left coronary segment was also thickened at its junction with the right coronary segment, to which it was adherent. It was, however, free from adhesion to the posterior segment, and, with the exception of the thickening of about one-third of its free edge, was healthy. The valves thus formed a thickened fringe surrounding the dilated aortic orifice. From the under-surface of the posterior segment the atheromatous degeneration extended downwards along the endocardium, lining the ventricle to the superior curtain of the mitral valve.

Half of the ventricular aspect of this valve was rough, its edges were thickened and studded with a few calcareous particles, and the tendinous cords attached to this portion of the valve were thickened and opaque. Some of the cords were very thick, and at their attachment to the valve were as large as crowquills. The anterior part of the valve was healthy. The auricular surface was smooth, but the edge of the valve was thickened and slightly rough towards the auricle. The other mitral curtain was not diseased. The valves on the right side presented a striking contrast to those of the left cavities on account of their healthy appearance. The aorta was smooth on its inner surface; its coats were thinned; but only one or two small atheromatous patches could be detected.

The following measurements were carefully made according to the plan mentioned by Dr. Peacock in his Croonian Lectures, with this exception, that the orifices were measured by circular discs instead of balls:

		Inches.
Length of the cavity of the right ventricle		5·25
„ „ left „		5·75
Thickness of walls of the right ventricle	base	·25
	midpoint	·31
	apex	·18
„ „ left „	base	·68
	midpoint	·62
	apex	·31
„ of the septum of ventricles		·5
Circumference of right aurico-ventricular aperture		5·5

	Inches.
Circumference of left aurico-ventricular aperture . .	4·75
„ aortic aperture	5·25
„ pulmonic „	3·87
Internal circumference of dilated aorta one inch above origin	6·25

The above measurements need no comment. The left ventricle, when opened and spread out so as to form a plane surface, measured half an inch below the origin of the aorta, 6·25 inches between its extreme limits, and in the middle of the cavity 7·5 inches. When dilated and distended with blood during life, the circumference of the cavity must have been much greater.

In the following observations I shall confine myself as much as possible to a consideration of the traces collected on the radial arteries by the sphygmograph, and those recorded by the apex-beat and the pulsation on the right side of the chest. The comparison of these with the lesions disclosed by the post-mortem examination form an interesting and by no means barren study.

The pulse-traces were verified by several observations. In their vertical line of ascent there was evidence of the hypertrophy of the left ventricle. In the little hook which caps each pulsation was the record of sudden ventricular contraction; and the fact that this hook was closely followed by the first secondary wave, causing a horizontal sum-

mit in the left trace, and a slight ascent in the right trace, indicated that the systole of the ventricle was long, and the heart muscle powerful enough to propel a full charge into the aorta.

The characteristics of free regurgitation into the ventricle are, a sudden fall in the line of descent of the pulse-trace after the hook-like summit, and effacement of the dicrotism. The absence of these features told us that the regurgitation was not great.

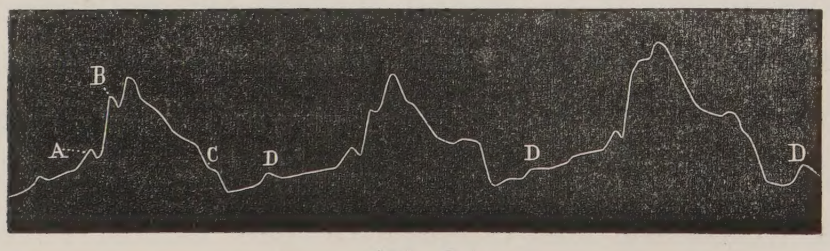
A consideration of the peculiarities common to both traces afforded this much information. A comparison of them, however, helped to another step in the diagnosis. The right pulse-trace was of much less amplitude than the left, was less vertical in the line of ascent, and differed also in the form of the summit. Differences of this kind, when constant, and when inexplicable by any local peculiarity affecting the circulation, always excite a suspicion of aortic aneurism. In this case some condition interfering with the equal distribution of the blood-current was rendered more probable by the diminished relative amplitude of the right pulse-trace, which is, as a rule, more ample than that of the left side. In the examination care was taken to exercise as nearly as possible the same pressure upon both the arteries. The index of the pressure-screw was adjusted at the same

point in both observations. The form and position of the aortic dilatation discovered after death were exactly calculated to interfere with the full force of the systolic wave, entering the brachio-cephalic trunk, while the vessels of the left side received without hindrance the blood directed towards them.

The post-mortem facts completely established the correctness of the view which the study of the pulse-traces had indicated during life. The case strongly supports the law which I am inclined to think further investigation will justify—viz., that aneurismal dilatation of the ascending part of the thoracic aorta produces diminution in the amplitude of the right radial pulse-trace.

The traces of the cardiac impulse and the movement at the point of pulsation on the right side of the thorax were obtained by means of the cardiograph.

With this instrument M. Marey has obtained the following trace, Fig. 3, which he has pub-



(Fig. 3.)

lished [1] as that of the normal cardiac impulse.

(1) Robin's *Journal de l'Anatomie et de la Physiologie*, No. 3, 1865.

In my own observations and experiments I have never succeeded in obtaining a normal trace so ample as the above. I have, however, been able to register traces of smaller amplitude possessing all the characteristics of the large trace. I have preferred to reproduce Marey's figure here because it shows more readily than a smaller one the several features of the heart-beat.

The interpretation of the trace is as follows.

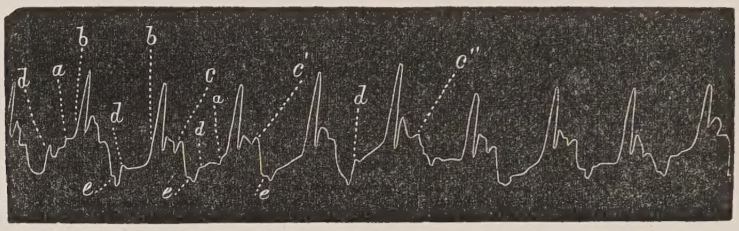
The trace of each cardiac pulsation commences with a slightly ascending line, in which the undulation marked A is produced by the true auricular systole. This is preceded by a smaller wave, marked D, in the second and third pulsations, which is caused by the entry of blood into the ventricle before the true systole of the auricle occurs. The line of ascent after A corresponds with the beginning of the ventricular systole: the first result of this is seen in the undulation B, which indicates the closure of the auriculo-ventricular valves. The period of the ventricular systole ends at C, which marks the closure of the aortic and pulmonic valves. The undulations which occur in the trace between B and C are vibratory in their nature and depend on oscillations following the closure of the mitral valves. The line of descent after C indicates the beginning of the ventricular diastole.

For convenience of reference, the trace of the

heart's impulse may be divided into an auricular and a ventricular portion. The auricular begins with the slightly ascending line of which D and A are undulations, and terminates immediately after the undulation A. The ventricular portion occupies the remainder of the trace. Modifications in the manner in which the ventricle is filled will affect the auricular portion of the trace. Modifications in the force and length of the ventricular systole, and in the mode of closure of the mitral and aortic valves, will be indicated by the height and amplitude of the ventricular portion, and by undulations in its summit, or towards its close.

Considering the heart-trace in reference to the pulse-trace, from A to the summit, corresponds with the line of ascent and summit wave of the pulse-trace; from the summit to C is represented in the pulse-trace by the first secondary wave, while the point C corresponds with the aortic notch. From C to A in the next pulsation is represented in the pulse-trace by the dicrotism and the remainder of the line of descent. In comparing the trace of the heart's impulse, Fig. 4, registered in the case under consideration, with the normal form, several peculiarities may be noticed. Beginning with the line of ascent, which corresponds to the filling of the ventricle after its contraction, we observe that the point *d*,

which corresponds with the elevation indicated by the same letter in the normal trace, shows a much more sudden elevation than in health. The height of this little wave is generally a test



(Fig. 4.)

of the perfect emptiness of the ventricle, and under such conditions is followed by a second strong wave in the line, corresponding to that marked A (Fig. 3). In the trace before us, however, we notice no relation of this kind; we have indeed but one well-marked sudden elevation, followed by the ordinary undulations which mark the line of ascent and correspond to A (Fig. 3). This sudden elevation of the pressure at the beginning of the filling of the ventricle indicates a considerable arrival of blood in the cavity, which comes partly from the auricle, but chiefly by regurgitation from the aorta. This peculiarity I have observed in several cases of aortic regurgitation, and the above is, I believe, its explanation.[2] The

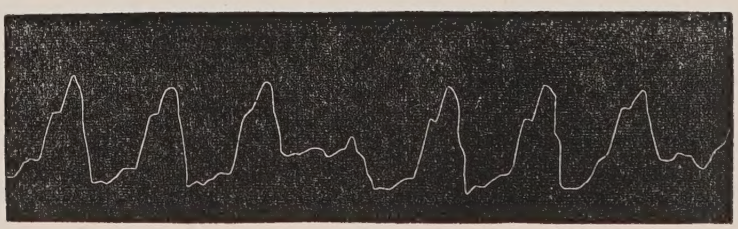
(2) This description of the cardiographic sign of aortic insufficiency was published in the *Medical Times and Gazette* of Sept. 26, 1866; in the *Gazette Médicale de Paris*, of Sept. 19, 1868, Marey published a paper describing this peculiarity of the heart-trace more at length, and calling

next peculiarity is the altitude of the line which marks the ventricular contraction, and by which the trace betrayed during life the hypertrophy and dilatation of the left ventricle discovered after death. The vibrations following this line are very fairly marked; but we notice that after the first fall of pressure, immediately succeeding the forcible systole of the heart, the line of the vibrations is almost horizontal in many of the pulsations, indicating the sustained vigour of the systole. The slight elevation at the end of the period of the systole, as seen in some of the pulsations (2nd, 3rd, and 4th), also points to the energetic character of the ventricular contraction. The closure of the sigmoid valves corresponds with the points marked *c*, *c′*, *c″*, but we also find near the end of the line of descent another indication of their closure at *e;* this may possibly be attributed to the vibrations of the valves caused by the backward flow of the blood into the left ventricle. In some cases of free aortic regurgitation, I have observed several undulations in the trace corresponding to the closure of the sigmoid valves; this can occur, however, only when the regurgitation is free, and consequently the tension in the aorta very low. The

attention to it as a new sign of aortic insufficiency. In the clinical remarks on cardiographic traces which follow Cases III and IV, I have shown, however, that it may be present in other valvular lesions.

line of descent in the trace (Fig. 4) has a vertical character, indicative of sudden diminution of pressure in the ventricle, which is not seen in the normal form. This is an indication of value in certain conditions, and here it tells us that the contraction of the ventricles was effectual, and completely emptied their cavities. In conditions which interfere with the complete expulsion of the blood, this line often has a more oblique form. The suddenness of the fall of pressure in this case was due to the diminished state of tension in the vessels, permitting the left ventricle to empty itself completely; and the obliquity which the right ventricle might have given to the line, if its pulsation could have been recorded, was masked by the greater share taken in the formation of the impulse by the hypertrophied left cavity.

The trace below, Fig. 5, was taken by applying the cardiograph over the point of pulsation between the second and third right costal cartilages.



(Fig. 5.)

A comparison of it with the heart's impulse and the trace of the radial arteries will suggest an ex-

planation of its principal features. The vigorous and prolonged ventricular contraction finds expression in the almost vertical upstroke and the altitude of the trace, while the sudden fall, scarcely broken by any indication of the closure of the aortic valves, corresponds with the diminution of dicrotism seen in the radial pulses. The peculiarity of the third pulsation is due to an inspiratory effort, the expansion of the chest-wall separating the bell of the cardiograph from the pulsation. In Fig. 3 we can also see the effect of inspiration on the cardiac impulse in the 3rd pulsation, and the same influence is seen to have modified the form of the 5th pulsation in Fig. 4.

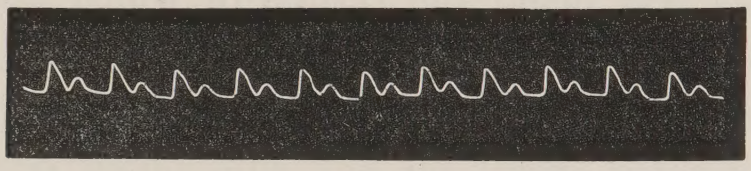
Case II.—*Adherent pericardium—Mitral insufficiency—Examination with the sphygmograph and cardiograph—Albuminuria—Dropsy—Death—Autopsy.*

William L., æt. 13, came under my care in August, 1866. The boy's mother stated that her son had always been delicate, but had suffered from no particular disease till October, 1865, when he was attacked by scarlatina, and subsequently by scarlatinal dropsy. Soon after the scarlatina, the heart symptoms first manifested themselves. When I first saw him his face was pale and puffy, his expression anxious, his lips a little dusky, and his breathing evidently quick. He coughed a

short dry cough from time to time, and complained that it harassed him constantly and broke his rest. There was a little mucus expectorated, but no blood; the ankles were slightly œdematous, and the face was remarked by his mother to be puffy every morning.

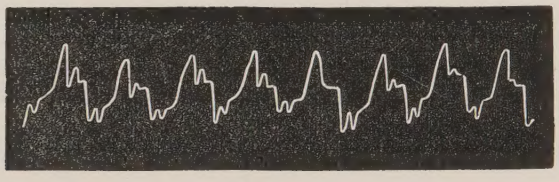
On examination the chest was seen to be ill-formed, rounded and prominent above, and flattened at the sides; over the heart it was flattened as if drawn inwards. On auscultation, the breathing was harsh all over the front of the chest; and posteriorly occasional rhonchi were heard. There was no pulmonary dulness on percussion either at front or back of chest. The respirations were thirty a minute. The cardiac dulness was increased laterally, extending from an inch beyond the nipple to the right edge of the sternum; the area of dulness was fixed, and did not vary with inspiration or expiration. The apex-beat was about two inches below and nearly one inch to the left of the nipple. The impulse was strong, wavy, and prolonged, producing a little movement of the chest wall, but ordinarily there was no sinking of the intercostal spaces with the systole. The first sound was accompanied by a loud blowing murmur, which lasted through the first pause, and was heard loudest at the apex. The murmur was well propagated towards the axilla, and could be heard all over the front of the chest, as well as

in the left vertebral groove. The second sound was inaudible at the apex, but was distinctly audible over the right ventricle; it was much intensified over the pulmonary artery, but was very feeble over the aorta. The pulse was 90, weak, and regular. The sphygmographic trace is seen below (Fig. 6). The cardiograph was applied, and



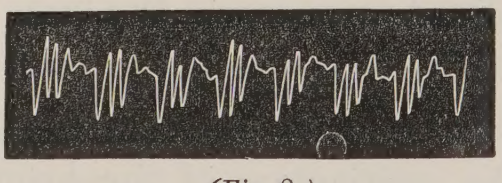
(Fig. 6.)

the trace below (Fig. 7) was obtained. While the



(Fig. 7.)

boy was under examination, an attack of palpitation came on, the heart beating very tumultuously; the apex seemed to strike the chest several times with each systole, and with the contraction some sinking in of the chest wall was noticed. Under these conditions the cardiograph recorded the following trace of the heart movement:



(Fig. 8.)

On examining the abdomen, the liver was slightly enlarged, there was no increase of splenic dulness; there was slight tenderness on pressure over the kidneys, and the urine contained albumen ($\frac{1}{3}$ col.) and numerous tube casts.

The sphygmograph and cardiograph were applied on several occasions, and a large number of observations was made, the results of which are well represented by Figs. 6 and 7. The striking features of the third trace, Fig. 8, were not repeated to the same extent on any subsequent occasion, but whenever the action was more excited than usual, there was a decided tendency to approach this type.

Under treatment the boy improved, and after a few weeks ceased to attend. During the severe winter weather, however, he became much worse; the dropsy returned, the heart symptoms greatly increased, and, an attack of bronchitis supervening, he died towards the end of January. No fresh inflammatory mischief connected with the heart occurred during his last illness. By the kindness of Dr. Mackey (under whose care the boy died, and by whom he was originally sent to me), I was present at the post-mortem examination. We were obliged to examine the body in the presence of his friends; on this account a thorough examination of the cavities could not be made. I append the particulars of the heart and great

vessels, to which we devoted our chief attention. The body, and especially the legs, were very œdematous, and the abdominal cavity contained a large quantity of fluid. On opening the thorax, the pericardium was found bound to the left side of the chest wall by means of greatly thickened and dense cellular tissue, and its parietal layer was everywhere adherent to the heart itself. When *in situ*, the heart, covered with the pericardium, measured 5·18 inches transversely and 6·25 inches vertically. The right cavities were full of black clotted blood, the left auricle also contained clots, but the left ventricle was nearly empty. The aortic and pulmonic valves were healthy. The superior curtain of the mitral valve was much thickened and evidently shortened; the tendinous cords were thick and opaque; the inferior curtain of the valve was only slightly affected. Small warty vegetations were scattered at intervals all round the circumference of the mitral aperture. The following measurements were made:

		Inches.
Length of the cavity of the right ventricle		3·56
„ „ left „		3·25
Thickness of the walls of the right ventricle	base	·25
	midpoint	·22
	apex	·13
„ „ left „	base	·75
	midpoint	·5
	apex	·43
Thickness of the septum of the ventricles		·5

Circumference of the right auriculo-ventricular aperture				5·25
„	left	„	„	3·
„	orifice of the pulmonary artery			2·25
„	„	aorta		1·87

The amount of the hypertrophy of the ventricular walls can be easily gathered from the foregoing measurements, which also indicate that the orifices of the pulmonary artery and the aorta were small when compared with the rest of the heart, and that the right auriculo-ventricular aperture was considerably dilated, while the left was but little altered, if anything it was contracted.

The diagnosis of general adhesion of the pericardium, especially when the history of the antecedent pericarditis is wanting, is by no means easy. In the case under notice, the signs pointed not very distinctly to the obliteration of the heart-bag, while the loud indication of the existence of mitral disease was too apt to withdraw the attention to itself as the only lesion. The peculiar absence of the second sound at the apex, however, particularly struck me, and, connecting it with the value assigned to this diagnostic by Aran,[3] I was led to seek more closely for other confirmatory evidence. The flattening of the side, the peculiarity of the impulse (coming, as it seemed to do, when the heart acted strongly, before the

(3) "Recherches sur les Adhérences Générales du Péricarde," *Arch. Génér. de Méd.*, Avril 1844.

first sound), and the unchanging area of cardiac dulness, all spoke strongly in support of the diagnosis of pericardial adhesion. The careful examination of the chest from time to time favoured this view, and particularly the observation of the heart's movements, for when the organ acted more violently than under ordinary circumstances, then the dragging of the chest wall was evident, and the diagnosis was strongly confirmed.

The sphymograph did not supply much information concerning the cardiac lesion. The vertical line of ascent, and the decided dicrotism in the pulse trace, told us that the blood stream entered the vessels quickly and forcibly, but that the arterial tension was nevertheless low. The regularity of the pulsations told us that we had to deal, not with a weak and flabby ventricle, sending the greater part of its contents back through a dilated mitral orifice, but rather with a strong muscular cavity, acting with energy, and not frustrated by a widely dilated mitral aperture favouring regurgitation.

The cardiograph, while it told us little of the mitral valve and its conditions, gave us pregnant indications concerning the movements of the heart. In the trace, Fig. 7, we see the two little undulations in the auricular portion which correspond in health with the filling of the ventricle. The first of these little waves in the trace before us

is very marked, and occurs almost immediately after the conclusion of the ventricular contraction —so soon after, indeed, that this alone excites some suspicion as to its connexion with the flow from the auricles. It must rather be referred, in my opinion, to the slight springing back of the chest wall after the action of the ventricles, the condition which produced the similar undulations in the trace Fig. 8. Throughout the trace we may remark, as in the pulse-trace, the signs of strong ventricular action, and of sudden and perfect closure of the sigmoid valves. In the trace (Fig. 8) these latter points are equally well marked, but we have in addition a striking record of the curious succession of beats against the chest wall which was felt with each systole of the ventricles when the heart was excited. These beats, when the hand was placed over the heart region, were strong enough to call away the attention from the true impulse, and forcible enough to register in the tracing an altitude greater than that caused by the real apex beat. At first sight these great waves preceding the elevation caused by the contraction of the ventricle, look like some marvellous intensification of the movements caused by the action of the auricles, but in some of the traces, and notably in the last two pulsations in Fig. 8, we see the little auricular undulation perfectly distinct from the preceding wave.

These two large waves, then, were due, not to any exaggeration of the auricular systole, not to any forcible and sudden distension of the ventricles, but to some other condition causing a movement forwards of the chest wall during the early part of the diastole of the ventricles. In the adhesion existing between the pericardium and the chest wall we have, I believe, the explanation of the fact. With each systole the wall was dragged upon by the heart muscle, and the recoil of the chest wall, and with it of the heart as soon as the ventricles were emptied, caused the movement registered by the instrument. This is a most important point, and, if my observation be confirmed, we shall have in this part of the trace a diagnostic sign of great value—the cardiographic sign of one form of adherent pericardium. Traube[4] and Friedreich[5] have already referred to the recoil of the chest wall after the ventricular contraction, but more especially when the pericardium had formed adhesions on its posterior aspect as well as anteriorly to the ribs. Potain[6] also some time before mentioned the distinct throb preceding the true impulse in cases of adherent pericardium. In the trace under our notice we see the experience of these observers confirmed by the record

(4) Zur Lehre von der Verwachsung des Herzens mit dem Herzbeutel, *Med. Zeitung des Vereins f. Heilkde. in Preussen*, No. 11, 1858.

(5) Virchow's *Handbuch der Pathologie und Therapie.*

(6) *Bullet. de la Soc. Anat. de Paris*, Août, 1856.

of an unerring instrument: we see the phenomenon, which they vaguely felt, telling its tale in no uncertain lines.

Case III.—*Aortic valve disease—Aneurism of the left subclavian artery—Examination with the sphygmograph and cardiograph—Gradual disappearance of the aneurismal tumour—Aggravation of heart symptoms—Asystoly—Death—Autopsy—Remarks.*

W. P., æt. 42, baker, was admitted into the Queen's Hospital, in December, 1865, under my colleague Dr. Fleming, by whom he was afterwards kindly transferred to my care. The man stated that he had suffered from severe pain in the left shoulder and arm, which he attributed to an injury sustained while carrying a sack of flour some five years before admission. On the occasion referred to he felt something give way in his shoulder, which caused faintness. He recovered in the course of half an hour, and thought no more of the circumstance. He never had any rheumatic attack, although he had been necessarily exposed to great changes of temperature in his work. About twelve months after the accident mentioned above, he began to be troubled with occasional pain at the heart and palpitation, and had since noticed his breath to be short on exertion. He had also suffered from indigestion. About

two months before admission to the hospital he had a cough and expectorated a small quantity of blood. The cough soon ceased, but he had lost flesh ever since.

When stripped for examination, his left arm and shoulder were found to be natural in size and shape. The chest was well formed. The percussion of the right side of the chest discovered no abnormal dulness, and the breath sounds over the right lung were natural. There was resonance all over the left lung except at the inner third of the clavicle, where the percussion note was dull. The respiratory murmur outside this spot was healthy. The dulness at the left apex extended downwards along the left edge of the sternum, and became continuous with the cardiac dulness, which reached from the third to the seventh rib, and laterally from the nipple line to the right edge of the sternum. The apex-beat was below and a little outside the nipple line, and was felt most distinctly between the sixth and seventh ribs.

On auscultation a loud double murmur was heard at the base of the heart. The systolic murmur was short and rough, the diastolic murmur was long and loud; both were heard most distinctly at the second right costal cartilage, but were distinctly audible all along the sternum and over the left clavicle. At the apex the systolic murmur

was very faintly heard, but the diastolic murmur was distinct, though less so than at the ensiform cartilage. In the left vertebral groove and in the left supra-spinous fossa both murmurs were heard, but not loudly. Above the left clavicle, under and outside the sterno-mastoid muscle, there was a pulsating tumour, as large as the base of a hen's egg. The tumour when grasped expanded freely, and on listening over it, two murmurs were audible, similar in character to those heard at the base of the heart. The right radial pulse was regular, jerky, and visible, beating eighty-eight a minute. The left radial was very weak and appeared retarded. The pulsations of the right radial, the carotids, and the brachials were distinctly visible. There were no indications of disease connected with the nervous or digestive systems. The pupils were equal. The liver and spleen were normal in size; the tongue was clean; the bowels were regular; and the urine was free from albumen. There was no dysphagia, and no great dyspnœa except on exertion.

The application of the sphygmograph to the

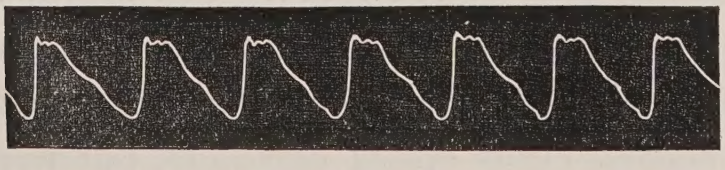


Fig. 9.—Right radial.

radial arteries gave the following traces (Figs.

9 and 10), which confirmed the previously made

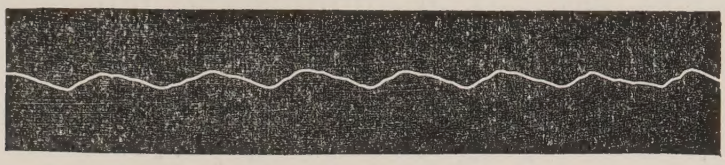


Fig. 10.—Left radial.

diagnosis of aneurism, and supported the opinion that the aneurism was not connected with the aorta, but only implicated the left subclavian artery. An aortic aneurism would not have affected the left radial artery so much more than the right. The cardiograph was applied to the pulsation in the neck and gave the following record (Fig. 11), which indicated the free expan-

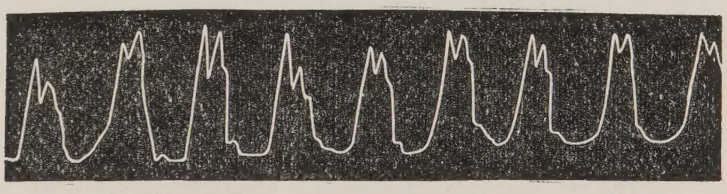


Fig. 11.—Aneurism.

sile movement of the sac at this time. Three weeks later the left radial pulse-trace became more perceptible to the touch, and gave the following trace (Fig. 12). This variation in the movements

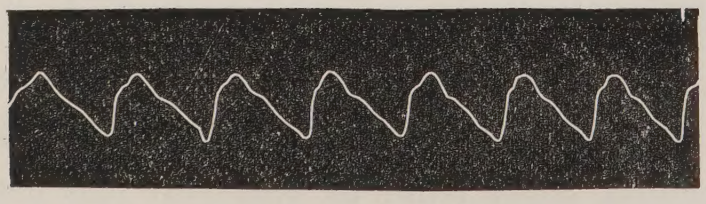


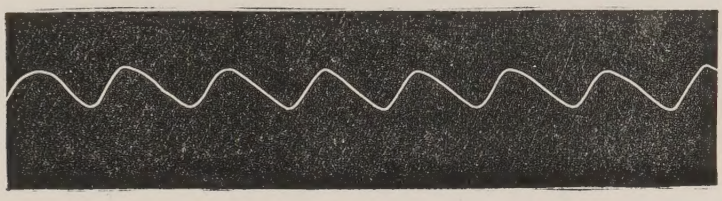
Fig. 12.—Left radial.

of the left radial artery was attributed at the

time to changes in the circulation through the aneurismal sac. The diminished pulsation and the hardening of the tumour which had been noticed for some days, rendered it probable that a deposition of fibrine in the interior of the aneurism had reduced its elasticity, and consequently diminished its modifying influence on the movement of the blood in the arteries beyond. The case had been treated by rest, restricted diet, and aconite internally, with anodyne applications to the arm and shoulder.

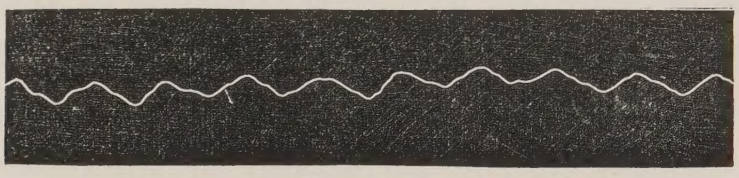
After a two months' stay in the hospital the patient requested his discharge, and afterwards came under my care at the General Dispensary. When leaving the hospital he fully understood the importance of the continuance of the treatment, and undertook to carry it out under my supervision. After some weeks' treatment at home, the tumour became less pulsatile, much more solid, and decreased in size. With these changes, however, there supervened great pain in the arm, shoulder, and left side of the neck. The arm had become much wasted, colder, and powerless. The fingers were cold and dusky-blue in colour, and shrivelled, and there was intense pain in the limb with starting of the muscles. Blisters also formed from time to time on the hand and fore-arm. The limb was wrapped in cotton wool and gentle friction used occasionally. For many

weeks this condition of the limb continued, and during this time the following traces were taken; they show the progressive diminution in the size of the left radial pulse. Fig. 13 is the trace soon



(*Fig.* 13.)

after he left the hospital. Fig. 14 was taken when



(*Fig.* 14.)

the pains and startings of the limb were very bad, the limb cold and almost pulseless, the brachial artery being very feeble and the radial almost imperceptible. At this date (August 19) the physical signs were as follows: At the heart apex the systolic murmur was indistinct, and the second sound replaced by the diastolic murmur, which increased in loudness towards the ensiform cartilage. At the base of the heart both murmurs were loud. The pulmonary second sound could be heard through the diastolic murmur at the left edge of the sternum. The murmurs were propagated upwards along the sternum and along

the innominate artery. On the left side they were also distinct over the tumour, but the systolic murmur was in this position harsher, longer, and louder than the diastolic. The heart's impulse was more forcible and diffused than formerly. The breath-sounds were everywhere natural, except under the left clavicle, where they were feeble and obscured by the murmurs.

The state of the arm and the condition of the aneurismal tumour, as described above, gave us at this time indications that the cure of the aneurism was being effected by the deposition of fibrine in its interior. The changes in the pulse-traces indicated the obstruction to the onward passage of blood caused by the formation of the clot. The pains in the limb, no longer like those which had formerly been present, when the tumour pressed more on the neighbouring parts, now depended on the scanty blood supply through the main artery, and the necessary formation of a collateral circulation. Four months later the tumour in the neck had become quite hard and solid, and was not much larger than a pigeon's egg. With the heart's systole it rose but did not expand. There was no murmur heard in it, but the cardiac murmurs could be heard conducted by the mass to the ear. The inner third of the left clavicle was dull, and the breathing under this clavicle was generally feeble. There was no pulsation in the brachial

artery, which could hardly be distinguished, and the left radial pulse was imperceptible. The sphygmograph, however, recorded the following trace (Fig. 15). The left arm was thin and all its

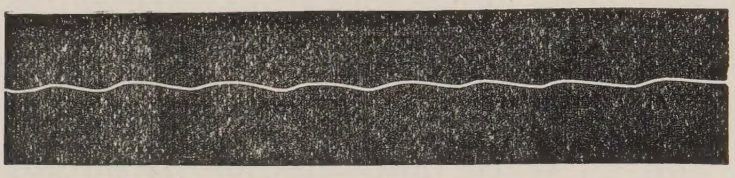


Fig. 15.—Left radial.

muscles wasted; he could only move it feebly, and regarded the limb as practically useless. It was colder to the touch than the right arm, but was not now so subject to pain, and was not so dusky as formerly. The cardiac conditions were unchanged. The carotids beat very visibly, and were equally forcible to the touch. The right radial was regular, large, and jerky; it gave the following trace (Fig. 16). At this time the man

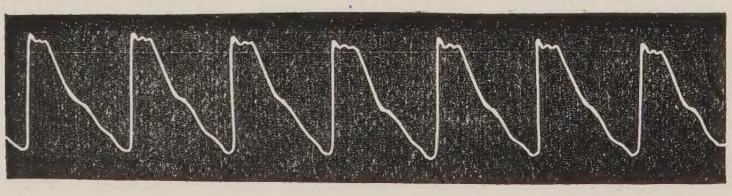


Fig. 16.—Right radial.

was much better in general health, and began to take some little exercise. He still had shortness of breath, but complained chiefly of the loss of power in his left arm. This defect made the cure of the aneurism seem to him a small gain. That it was cured I concluded from the state of

the tumour, the characters of the pulse-trace obtained on the left radial artery, and the stages through which the limb had passed. The left radial pulse-trace (Fig. 15) I regarded as the product of the blood movement from the establishment of the collateral circulation.

Unfortunately the man's circumstances compelled him to work, and, failing to obtain other employment, he returned early in the year 1867 to his former occupation as a baker. A few weeks' work sufficed to lay him up with shortness of breath, cough, œdema of the legs, swelling of the abdomen, and great cardiac distress. I saw him on the day before he died, and found him propped up in bed, breathing very rapidly. His face was puffy, his lips were blue, the breath was coldish, and there was constant sickness. The temperature in the left axilla was only 94·6.° The veins of his neck were turgid, the heart action was confused and labouring, and the aortic murmurs could no longer be distinguished clearly. A mitral systolic murmur was heard at times very distinctly. The area of cardiac dulness was greatly increased laterally, extending one inch beyond the right edge of the sternum. The left pulse was imperceptible, the right was feeble and intermittent. The breathing over the superior part of the chest was loud and harsh. Both lung-bases were dull posteriorly, and liquid subcrepi-

tant rhonchi were heard all over the back of the chest. The liver was enlarged, reaching more than one inch below the costal arch. The urine was high-coloured, scanty, and albuminous. In spite of treatment, he died a few hours after I saw him. Permission was obtained to make an examination of the chest the next day, and the following were the results.

The left arm was found to be greatly wasted. The tumour in the neck could be felt as a hard lump beneath the sterno-mastoid muscle, not larger than a small walnut. On opening the thorax the heart was seen to be very large, and its right cavities were distended with blood. There were several ounces of fluid in the pericardium, and about half a pint in each pleural cavity. The lungs were congested and œdematous. The heart and great vessels were removed, and with them the left subclavian artery beyond its termination. The right ventricle was found to be greatly dilated, but its valves were healthy. The left ventricle was hypertrophied and greatly dilated. The muscular fibre was rather soft and in parts pale. The musculi-papillares of the mitral valve also showed fatty change in spots. The mitral valve was thickened, especially its superior curtain, which was also shortened and undergoing degenerative change. The aortic valves were incompetent. The in-

competence was due to thickening and shortening of all the segments, but especially to retroversion of the posterior segment, which was torn down at its attachment to the right coronary segment to the extent of $\frac{1}{6}$th inch. The valvular segments were all thickened, and in spots undergoing calcareous degeneration. The aorta was dilated, but not greatly so; was rough, inelastic, and studded with cartilaginous and calcareous patches of atheromatous change. The degeneration affected the whole of the arch of the aorta, and to a less degree the innominate artery. The left carotid was fairly healthy, but slightly larger than natural; the mouth of the left subclavian was narrowed and surrounded by atheromatous change. This vessel was pervious for about one inch above its origin, and from that point was filled by a hard clot of dense fibrine. Beyond the scalenus anticus the artery was very small and wasted. The dilatation had evidently been greatest at the curved portion of the artery which is internal to the scalenus muscle. In this situation the fusiform dilatation became more globular, especially on the upper side. The clot in this part was larger and extended into the branches of the vessel for a short distance. The vessel was connected to the pleura by old dense inflammatory adhesions.

Apart from its interest as a case of aneurism

which had undergone cure, the preceding case presents very clear evidence of the value of the sphygmograph in diagnosis. The comparison of the differences between the right and left radial pulse-traces clearly indicated the left subclavian artery as the diseased vessel. Even the most developed traces collected on the left radial differed too greatly from the right radial trace to make the diagnosis for a moment uncertain. An aneurismal sac in the course of an artery produces much greater changes in the blood movement in the vessels beyond it, than an aortic aneurism. These changes are directed to the blending of the three distinct parts of the normal trace, and their confusion in a single curve; thus in the traces collected in this case below the aneurism, we notice that the line of ascent and summit wave are always modified, and the dicrotic wave still seen in the right trace, is further diminished or lost in the left; the part of the trace least affected being the first secondary wave or pressure wave. This formed the summit of the trace on the left side, and thus the pulse lacking the true vibratory summit wave felt on the right side, and having its pressure wave slightly delayed, was perceived by the finger to be retarded.

The modifications in the line of ascent and the form of the summit wave are commonly the most important signs in the pulse-trace indicative of

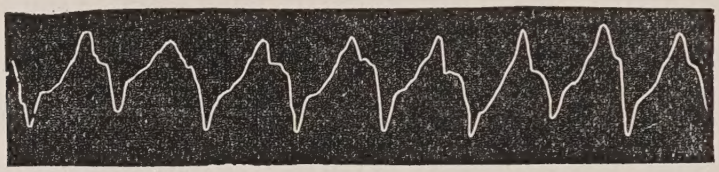
aneurism. They are perceived by the finger as a loss of force in the radial pulse of the side affected. The modifications of dicrotism are not perceived by the finger readily, but often render in the sphygmographic trace important diagnostic evidence. This is especially so in some cases in which the existence of a large aneurism exaggerates by its contraction the dicrotic wave, or adds supplementary waves to the line of descent.

The pulse traces (Figs. 9 and 16) collected on the right radial deserve attention also, not only on account of the contrast offered to the left radial pulse traces in the vertical line of ascent, the small but distinct summit wave, and the slight dicrotic wave, but also on account of the information given by these features as to the nature of the aortic valve mischief. The almost flattened summit in these right traces indicated the existence of considerable disease of the aorta (endarteritis deformans), and suggested the view that the valvular mischief and the aneurism were secondary conditions. The man's occupation necessitated the carrying of heavy weights, and in this way great strain was thrown upon the aorta at times. This no doubt gave rise to the inflammation of the inner coat, which, extending downwards, implicated the valves at the mouth of the aorta, and finally led to the retroversion of the segment, which was found torn after death. From

the moment the accident occurred the necessity for ventricular hypertrophy was increased, and as the left ventricle grew stronger and propelled a large charge of blood into the aorta at each systole, so the strain on this vessel was aggravated and the inflammatory changes in its coats were stimulated. A vicious pathological circle was established. Under these circumstances the left subclavian artery became affected first with inflammatory change, and later on with the aneurismal dilatation of its coats. Such was the theory of the case which the study of the pulse traces suggested: we have seen that the autopsy confirmed it.

The explanation of the varying forms of the left radial is to be found in the gradual obliteration of the subclavian artery, and the last trace (Fig. 15) may be regarded as the sphygmographic evidence of a feeble circulation in the radial from collateral sources. The trace collected on the tumour (Fig. 11) when pulsating most vigorously gave an indication of a double beat which could be felt indistinctly by the hand. The two waves correspond to the summit wave and the first secondary wave of the right radial pulse, the vibratory element or summit wave being especially well marked from the proximity of the sac to the aorta and the low state of arterial tension which the incompetency of the aortic valves produced. At

the bottom of each pulsation a slight indication of the dicrotic wave is recorded. On several occasions the heart-trace was taken by the cardiograph, and the trace below (Fig. 17) represents



(Fig. 17.)

the form of the apex beat. It presents several noteworthy features. In the sudden ascent of the auricular portion of the trace, at the beginning of each pulsation, we see the indication of the filling of the ventricular cavity by the reflux of blood from the aorta. The normal auricular waves are almost lost in this abnormal elevation. A second feature worthy of notice is the feeble indication of the closure of the mitral valves. There is, indeed, only a slight break in the line of ascent. This is no doubt due to the abnormal conditions of the closure of the mitral valve curtains. When the systole occurred the closure of the valve had been partially effected by the reflux of blood into the ventricle, and consequently the mitral undulation of the normal trace was blended in the upstroke of the ventricular systole. This is, I believe, the explanation of this peculiarity, as it is of the absence of any clearly marked first sound in some cases of aortic regurgitation.

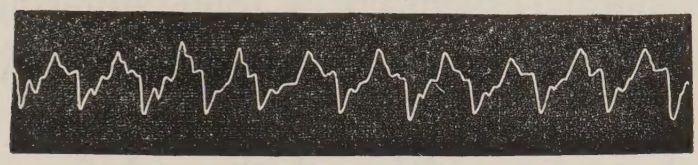
The sudden fall in the line of descent of the trace after the point indicating the closure of the aortic valves, is another deviation from the normal form which is often seen when the heart muscle is hypertrophied and the contraction sudden. In some of the pulsations there occur also undulations in the line of descent similar to those observed in the trace Fig. 4, p. 280, and which may be referred to vibrations produced at the aortic valves by the regurgitant blood current.

It may not be out of place here to show that the sudden elevation at the beginning of the auricular portion of the heart-trace may be present in conditions other than aortic regurgitation. I have already pointed out (pages 285, 290-1) how remarkably this peculiarity was developed in a case of adherent pericardium associated with mitral regurgitation. In that case the abnormal elevations were explicable by the special pathological conditions which the post-mortem examination disclosed. In health, vigorous contraction of the ventricle favours the production of a slight elevation in the trace, by rendering the cavity so completely empty that blood rushes in quickly from the auricle. So in disease, any conditions which insure perfect expulsion of each charge of blood from the ventricle, and at the same time cause the blood to accumulate under high pres-

sure in the auricle, may be regarded as favourable to the sudden filling of the ventricle which this peculiarity indicates. We have seen how, after vigorous ventricular action in aortic valve disease, the sudden reflux of blood from the aorta will produce this abnormal elevation. Under strong muscular effort, the same effect is caused in a minor degree, from the copious blood-wave rushing in from the left auricle. In mitral regurgitant disease we have a pathological state which favours the over-distension of the auricle, and so renders it ever ready to pour a full wave into the ventricle as soon as its diastole begins.

In order to test this view I collected a number of heart-traces from cases of mitral regurgitant disease. My first observations were made on a boy æt. 16, whom I had treated for rheumatic fever and endocarditis some six months previously. He made a good recovery from the rheumatic attack, but a loud blowing systolic murmur remained. This murmur was loudest at the apex of the heart, was propagated towards the axilla, and was very distinctly heard posteriorly at the angle of the left scapula. At the base of the heart the murmur was not very distinct. The pulmonary second sound was accentuated. The apex-beat was sharp and forcible between the fifth and sixth ribs, and as his chest wall was thin, the cardiograph collected a good trace. The following

Fig. 18, is a typical specimen of the numerous



(Fig. 18.*)*

traces recorded in this case. The sudden rise at the commencement of the auricular portion shows how suddenly the blood entered the ventricle from the distended auricle. The small wave which follows is a well-marked record of the vigorous contraction of the auricle itself. The ventricular portion is characteristic of a prolonged and strong systole, in which the vibrations of the mitral valve are well expressed. The point of closure of the aortic valves is sharp and single, indicating sound valves and a strong ventricle. The sudden fall in the trace which follows shows how thoroughly the ventricle emptied itself, and consequently how fit it was to receive an abundant wave from the auricle as soon as its walls relaxed. The auricle was always ready with this wave, and so each pulsation begins by the sudden elevation of the line of ascent.

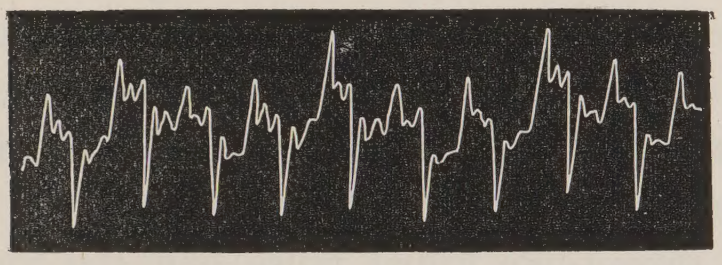
In the following case in which mitral regurgitation was the principal lesion, these peculiarities of the heart-beat were even more developed.

Case IV. — *Mitral insufficiency — Examination with the cardiograph—Death—Autopsy.*

J. R., æt. 28, bookbinder, came under my care at the General Dispensary in 1866, suffering from pain at the heart, palpitation, shortness of breath, cough, and œdema of the feet. She was a tall, well-made woman, with a dusky red face, bluish lips, and anxious expression. She had never had any illness except rheumatic fever, of which she had a severe attack when thirteen years of age. The heart symptoms had only come on for the past five years, and rendered it very difficult for her to undergo any exertion. A short walk of a quarter of a mile to the Dispensary was observed to raise her breathing to thirty a minute. The pulse was always rapid and regular, but very small. There was nothing abnormal in the lungs beyond some moist sounds posteriorly at the bases. The liver was slightly enlarged, the urine was scanty and high coloured, but free from albumen. The chest was well shaped and thin, so that the heart's impulse could be distinctly seen. There was heaving all over the cardiac region with the systole of the ventricles, but the apex-beat was sharply and distinctly felt one inch and a half vertically below the nipple. The dulness extended laterally to the right edge of the sternum. On auscultation a soft blowing, almost musical murmur, was heard with the first sound at the apex, and was followed by an accentuated second sound. The murmur was heard at the base, and was

propagated all over the front and back of the chest. The second sound was clear at both base and apex, but strongly accentuated over the pulmonary artery.

The patient was frequently under treatment for three years, and was repeatedly examined with the sphygmograph and cardiograph. The heart-trace is figured below (Fig. 19). She had several



(Fig. 19)

bad attacks of pulmonary apoplexy, and finally died in the General Hospital under my care. At the post-mortem examination the pericardium was found to contain a few ounces of serum, and on the anterior surface of the heart there was a well-defined white spot, which would have been referred to the accoutrements, if it had occurred in a soldier, instead of a woman.

The heart weighed seventeen ounces, was globular in shape, and greatly hypertrophied. The right cavities were dilated, and the walls of the right ventricle were thickened. The valves and orifices on the right side were healthy. The left auricle contained, in addition to black blood, a

large fibrinous clot. Its walls were thick and its cavity greatly dilated. The left ventricle was greatly hypertrophied. The aortic valves were competent. The mitral valves were much diseased and were incompetent. The two curtains were thickened and adherent, and thus formed a somewhat rigid aperture which admitted two fingers as far as the first joint. The aperture was rough and studded with irregular hard vegetations. The other viscera presented the usual appearances found after old mitral regurgitant disease.

One of the most striking features of this case during life was the contrast between the small, weak, thread-like radial pulse and the vigorous heart-throb. There was evidently some condition which robbed the artery of its due supply of blood, and the permanently patent condition of the mitral valve found after death explained the peculiarity. The conditions were most favourable to test the value of the indications of the cardiographic trace. The distended auricle must have poured a full wave into the ventricle as soon as the ventricular diastole began, and during the whole of the ventricular systole the tense but incompetent mitral valve curtains must have oscillated as the blood current rushed back between them into the auricle. The trace of the heart-beat, Fig. 19, which is a typical specimen of the form in this case when the heart was acting

vigorously, shows features characteristic of these conditions.

The cardiographic records in these cases are, I think, sufficient to show that the sudden elevation in the auricular portion of the heart-trace may be caused by mitral regurgitation, and that, therefore, it is not characteristic of disease of the aortic valves.

When it is recorded in a case of mitral valve disease, it indicates the existence of an easy and rapid passage of blood from the auricle to the ventricle, such as insufficiency of the mitral valve allows. On the other hand, when the auriculo-ventricular aperture is narrowed by adhesions of the curtains of the mitral valve the conditions are altered: the passage of blood into the ventricle from the auricle is no longer easy and rapid, but difficult and slow, and consequently the sudden elevation in the auricular portion of the trace is replaced by a gradual rise. This is well illustrated by the following case.

Case V. — *Mitral obstructive disease (mitral stenosis)—Præsystolic murmur—Examination with cardiograph.*

S. B., æt. 20, sempstress, came under my care at the Queen's Hospital, in June, 1866, complaining of shortness of breath, palpitation, and cough. She was a pale, anxious-looking woman, marked

with the small pox. She had never had any serious illness since she was a child, when the attack of small pox occurred, but for some years past had suffered from a constant, dry cough, great dyspnœa on exertion, and occasional faintness. On some occasions, when her breath had been worse, she had expectorated blood. Her appetite was good, her tongue was clean and red, the bowels were regular. There was no œdema of the feet. The pulse was regular, but small, beating eighty times a minute when she was at rest, but becoming much more rapid and still smaller on the least exertion. The sphygmograph recorded a trace normal in form but of low tension, and with some beats smaller in amplitude than others, indicating a variation in the amount of blood discharged at each ventricular systole.

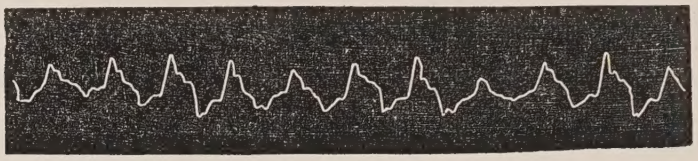
On examination the lungs presented no marked signs of disease, nor was there anything specially noteworthy in the condition of the abdominal organs. The urine was free from albumen. The area of cardiac dulness was increased, extending laterally to, or slightly beyond, the right edge of the sternum, and from the third rib to the sixth interspace. The apex was seen to beat between the sixth and seventh ribs, and the hand perceived that it was preceded by a thrill. At the base of the heart the sounds were clear, but the

pulmonary second sound was strongly accentuated. At the apex a harsh blowing murmur was heard preceding and running up to the first sound, which was flapping in its character. When the hand was placed over the apex-beat the murmur could be heard while the thrill was felt, and both could be perceived to finish with the true impulse, and immediately before the flapping first sound. The murmur was also noticed to precede, and finish before, the carotid and radial pulse beats.

The murmur thus made out to be clearly præsystolic was not heard beyond the area of the apex. When the frequency of the heart's action had been reduced by digitalis, the præsystolic character of the murmur was so easily made out that the case served excellently for class demonstrations. This patient remained under observation for some months, and was repeatedly examined with the sphygmograph and cardiograph. The pulse-trace always retained the characters mentioned above, but indicated an improved state of tension when the heart's action was slow under the influence of digitalis. The trace of the apex-beat (Fig. 20) is a typical specimen of the form observed.

There is in this trace no sudden elevation of the auricular portion following immediately after the line of descent. There is, on the contrary, a

gradual but decided rise of the line of ascent all through the auricular portion, in which the true



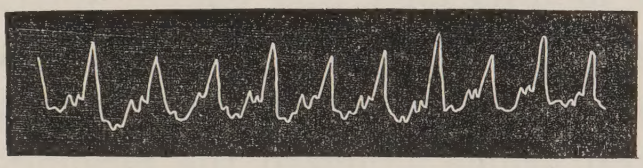
(Fig. 20.)

auricular wave is generally absorbed. When the wave is present, however, its relatively large size shows how vigorously the auricle contracted. In the curved line of descent, after the closure of the aortic valves in some of the pulsations, is seen the influence of the over full right ventricle emptying itself, but slowly, on account of the increased tension in the pulmonary artery. The ventricular portion indicates a comparatively short and vigorous systole, unbroken by the oscillations seen in mitral regurgitant disease.

This trace bears the special characters, which the form of disease would have suggested on *a priori* grounds. The only point in which it fails is in the record of the præsystolic thrill; in other cases a series of small waves in the auricular portion of the trace (*vide* Fig. 21) have represented the thrill perceived by the hand. I have recorded this case here, because it was one of a series used to illustrate the various modifications in the heart trace, in a paper read before the Birmingham

Branch of the British Medical Association in February, 1867.[7]

In the following case of mitral stenosis the auricular portion of the heart-trace showed well-marked undulations corresponding with the præ-systolic thrill felt by the hand. These are well represented in the trace (Fig. 21) which was col-



(Fig. 21.)

lected from the heart-beat of a young woman, aged sixteen, who came under my care early in 1868. She was a pale and delicate-looking girl, who suffered from palpitation, dyspnœa, cough, and œdema of the feet and legs. Four years previously she had had an attack of rheumatic fever, during which she was blistered over the heart. The pulse was generally regular, but yielded a trace indicating low arterial tension. On auscultation at the left apex a rather harsh murmur was heard, preceding, running up to, and ending abruptly with, the first sound. There was no murmur audible after the impulse, or the carotid pulse, was felt. The murmur was distinctly præ-systolic, was not propagated towards the axilla,

(7) Vide *British Medical Journal, March 16th,* 1867.

and was not heard over the right ventricle or at the back of the chest. Both sounds at the base were clear; the pulmonic second sound was intensified. The apex-beat situated between the fifth and sixth ribs was forcible and was preceded by a distinct thrill, which ended in the true impulse. The area of cardiac dulness was normal. Some eighteen months later this patient died; and on post-mortem examination, the mitral valve curtains were found to be adherent and thickened. The mitral orifice admitted the forefinger as far as the middle joint, and the circumference of it on the auricular surface was roughened by spots of calcareous deposits. The valve, although thick, was not rigid. The other heart valves were healthy.

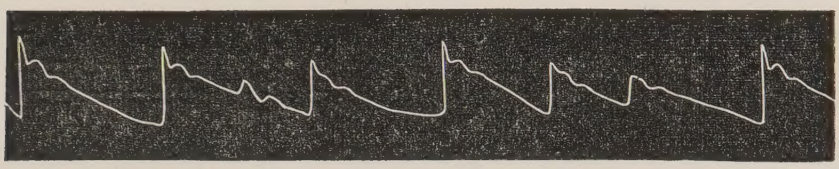
In both the preceding cases of mitral stenosis the heart action was regular, and the murmurs were loud, rough, and rather prolonged. These features are by no means constant in this form of valvular mischief. The pulse, on the contrary, frequently exhibits a very characteristic irregularity, and the murmur may be soft, short, puffing, and strictly limited to the moment immediately preceding the ventricular systole. Sometimes, indeed, the murmur may be lost, and again be heard after a time. The following case illustrates these points.

Case VI.—*Mitral obstructive disease—(mitral stenosis)—Præsystolic murmur—Examination with the sphygmograph—Death—Autopsy.*

G. C., æt. 38, labourer, came into hospital under my care in May, 1868. Some twelve years previously he had suffered from rheumatic fever, but till the last two years he had been in good health. For some time prior to his admission he had complained of pain in the epigastrium, dyspnœa, and palpitation. The symptoms increased in intensity, his legs and feet began to swell, and he had a troublesome cough accompanied by bloody expectoration. When he was admitted the dyspnœa was very urgent, the lips were blue, the skin and conjunctivæ yellowish, and the expectoration bloody. The liver was enlarged, reaching two inches below the costal arch. The urine was free from albumen. The pulse was small, weak, and irregular. The percussion note was impaired over both lung bases, and thin, liquid rhonchi were audible at the left base. The area of cardiac dulness extended from the left nipple line to the right edge of the sternum, and from the third rib to the sixth space. The impulse was heaving, diffused, and visible in the epigastrium. On auscultation, the irregularity of the heart's action made the definition of the sounds difficult; a faint blowing murmur was heard, however, at the left

apex, preceding the first sound. There was no murmur audible at the base with either sound, or in the axilla. Under treatment, in which digitalis was the chief drug administered, the man improved greatly. The pulse became firmer, slower, and more regular; the œdema disappeared, and the other symptoms decreased. In this improved state of the patient the murmur was observed to vary greatly in intensity. Sometimes it was very faint, at others it was absent, and again, especially after exertion, it could be detected easily. When most distinct, it was audible only at the apex, and was not propagated upwards or towards the axilla. A crown-piece fully covered the whole area over which it was heard. It was a soft sound, like a puff, commencing after the middle of the long pause, and ending abruptly with the first sound, which was short and flapping. The murmur preceded the heart's impulse and the carotid pulse. At the base of the heart both sounds were clear; the pulmonic second sound was louder than the aortic. A large number of observations were made with the sphygmograph, and the following trace (Fig. 22) is a typical specimen of the pulse-form in this case when the urgent symptoms had subsided. It shows by the unequal amplitude of the pulsations the varying quantities of blood sent into the arteries by the vigorous ventricular contractions. The ir-

regularity of the heart's action is shown by the widely different intervals at which the pulsations



(Fig. 22.)

succeed one another. The occasional appearance of a small abortive pulsation in the line of descent, as seen in the second pulsation, is a special characteristic of this form of valvular disease. My observations lead me to conclude that it is due to a premature auricular contraction, which propagates itself to the ventricle, and which is caused by the increased blood-pressure in the auricle associated with certain phases of the respiratory act. This abortive pulsation sometimes looks like an exaggeration of the dicrotic wave, which in reality was singularly absent from the trace in this case.

Ten weeks after his first admission this patient was again brought to the hospital moribund. The post-mortem examination discovered extensive recent apoplexy of the left lung, œdema of the right lung, and effusion into both pleural cavities. The heart was large and globular in shape, and weighed seventeen ounces. The right cavities were greatly dilated, and the right

ventricle hypertrophied. The valves of the right side were healthy, but the right auriculo-ventricular orifice was narrower than in health, and admitted only the two first fingers freely. The left auricle contained black blood clots, was dilated, and its walls were thickened, measuring in many places $\frac{3}{16}$ inch in thickness; the endocardium lining this cavity was also thickened. The left ventricle was not dilated nor hypertrophied, except as regards the papillary muscles which were thicker than natural, their tendinous chords being shortened. The curtains of the mitral valves were adherent and formed a funnel-shaped passage into the ventricle which ended in a button-hole aperture, into which the top of the forefinger only could be passed. The sides of the aperture were smooth, and its circumference and the walls of the funnel, although thickened, were not rigid, so that, when the valve fell back under the pressure of fluid in the ventricle, its sides flapped together, and the passage to the auricles was closed.

The peculiarities of the murmur in this case, and the special features of the pulse-trace are those which I have commonly found present in cases of mitral stenosis, produced by funnel-shaped adhesion of the mitral valve. In the earlier stages of these cases the pulse, though of low tension, is often regular, and the mur-

mur is frequently very faint. When the mitral orifice is less narrowed, but more roughened, the murmur is louder and more prolonged, and the pulse often regular. In some cases I have noticed that, as the degenerative changes in the valve curtains advance, a double mitral murmur may be detected, indicative of the development of secondary mitral insufficiency.

The physical signs of mitral stenosis have now become so generally well known, and the value of the præsystolic murmur so thoroughly established as the sign of this lesion, that it is unnecessary to illustrate this form of heart disease by more cases. The corresponding lesion on the right side of the heart is, however, the rarest of all valvular affections, and it has fallen to the lot of few physicians to observe a case. So rare is it that the great master of auscultation, Skoda, has declared that he would incline to regard a diastolic murmur heard over the tricuspid valves as pericardial, rather than valvular, in its origin. Tricuspid stenosis is, however, met with in the post-mortem room. I have myself seen three specimens of it associated with a similar but more developed condition on the left side of the heart, and in one instance a tricuspid murmur was detected in addition to the mitral murmur. In the following case, which I observed carefully during life, the tricuspid valves were practically alone

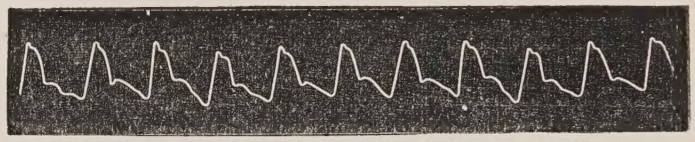
diseased, and gave rise to the physical signs which led to the diagnosis of the lesion.

Case VII.—*Tricuspid obstructive disease (tricuspid stenonis)—Præsystolic murmur—Examination with the sphygmograph—Death—Autopsy.*

T. S., æt. 35, striker, came into hospital in the latter part of 1868 suffering from great difficulty of breathing and palpitation, associated with a constant aching pain at the ensiform cartilage. He referred his illness to an attack of rheumatic fever which had occurred six years previously, and had disabled him more or less ever since. For months before his admission he had suffered from occasional attacks of epistaxis, disturbed sleep, breathlessness on exertion, palpitation, and pain at the ensiform cartilage. His face after he was admitted was noted to be puffy, and dusky in colour; his lips were slightly blue. The jugular veins were distended, and a slight undulatory pulsation was observed in the right vein, occurring prior to the heart's systole. The feet were slightly œdematous; the urine was free from albumen, and the bowels were regular. On examination the lungs were found to be normal on percussion, and the breath sounds healthy. The area of cardiac dulness was increased laterally, reaching from the nipple line to half an inch beyond the right edge of the sternum. The apex was felt below the sixth

rib, but the epigastric pulsation was unusually distinct. When the hand was placed across the front of the chest over the heart, a distinct thrill was felt, which was most distinct at the ensiform cartilage. The thrill appeared to precede the true impulse. On auscultation at the base the first sound was clear, and the second sound, though less distinct than usual, was free from murmur. It was followed, however, by a faintly heard murmur, which ran up to and ended with the next first sound. This murmur grew louder as the ensiform cartilage was approached, and had its point of maximum intensity at the base of the ensiform cartilage close to the right edge of the sternum. It began softly, but grew louder and harsher up to its end, which came abruptly with the first sound. It grew fainter as the left apex was approached, and at the apex-beat the two normal sounds could be heard nearly free from the murmur. The murmur was propagated faintly to the base of the heart, but was not audible in the axilla or at the back of the chest. These particulars were made out by repeated examinations, for the situation of the murmur and its direction of propagation were so unusual that they excited much interest. The question of its origin was much discussed. Was it an aortic regurgitant murmur? Was it pericardial? Was it the præsystolic murmur of mitral stenosis propagated unusually?

All these suggestions were canvassed, and one after another dismissed. The pulse-trace (Fig. 23)



(Fig. 23.)

gave no evidence of aortic regurgitation; and the well-marked dicrotism, indeed, contradicted this view. Moreover, the murmur was not heard along the great vessels; on the contrary, a well-marked second sound was detected in the carotids. The murmur differed from an aortic murmur also in growing louder and harsher towards its end, and in commencing, not with the second sound, but distinctly after it. It was not propagated in the direction of a pulmonic regurgitant murmur. The mitral stenosis hypothesis was contradicted by the area over which the murmur was heard, by its absence over the mitral valve, by the non-accentuation of the pulmonic second sound, and by the freedom of the patient from those lung complications which would have been the inevitable accompaniment of the dyspnœa, the congested face, the blue lips, and the turgid jugulars, if these phenomena had depended on disease of the left side of the heart. The cyanosis throughout was remarkably out of proportion to the amount of lung trouble. A pericardial origin of the murmur was con-

sidered, but after long consideration was dismissed, and the diagnosis of tricuspid stenosis was made. For three weeks after his admission the man improved, and the physical signs were studied from day to day with the result of confirming the opinion as to the origin of the murmur. At the end of the third week the patient caught a severe cold from imprudent exposure in leaving his ward, and was laid up with severe bronchitic symptoms. After a few days his state improved, and feeling better he got up, contrary to orders. An hour or so afterwards he was seized with severe rigors, great dyspnœa, intense pain at the ensiform cartilage, great lividity of the surface, and sweating. He was freely cupped, and had stimulants given internally, but died the next day. For some hours before death his radial pulses became very weak, and the left pulse was the first to become imperceptible.

On post-mortem examination old pleuritic adhesions were found on both sides, the lungs were congested and the tubes were full of frothy mucus. The heart was seen to encroach considerably on the right side, the right auricle being greatly distended. The pericardium was adherent to the front of the heart and over the situation of the white spot, the adhesion was old, and cartilaginous in consistence for a space of about one-third of an inch in diameter. The left cavities contained

little blood. The left ventricle was slightly hypertrophied. The right ventricle was full of blood, but was not dilated, nor were its walls thickened. The right auricle was greatly distended and its walls were hypertrophied, the endocardium was tough, opaque, and thick, the walls being in many parts one-quarter of an inch in thickness. The tricuspid valve segments were adherent, and formed a round orifice of communication between the auricle and ventricle, which admitted the forefinger beyond the first joint. The edges of the orifice were thick and rough on the auricular surface and studded with calcareous spots. The aortic, pulmonic, and mitral valves were healthy. The orifice of the aorta, and the left auriculo-ventricular aperture were narrowed, the latter only admitting the tips of the two first fingers. The liver was small and granular. The spleen was large and congested. The kidneys were congested, but in other respects healthy. The peritoneal cavity contained a little more than a quart of serum.

Hitherto a præsystolic murmur caused by tricuspid stenosis, has been regarded by most writers on heart disease as a theoretic possibility. The preceding case removes the murmur from the category of possibilities and places it in the class of observed facts. Some two years after my case had been read and the specimen exhibited

at the Birmingham Medical Societies, Dr. Hayden published [8] a case in which, among other valvular lesions, constriction and inadequacy of the tricuspid valves were present, and had produced a double tricuspid murmur during life. The case is of much interest and worthy of reference, although the many murmurs present and the complex valvular lesions, diminish its value as evidence of the pathognomonic sign of tricuspid stenosis.

Dr. Gairdner has recorded [9] a case in which a murmur, beginning after the second sound, continuing throughout the pause, and going on up to the first sound, at which it stopped abruptly, was referred to tricuspid obstruction. The patient died in 1872, and the accuracy of the Glasgow professor's diagnosis was verified by the discovery of obstruction at the tricuspid orifice, due not to any valvular flaw, but to "a tumour attached to the auricle" which "floated down upon the orifice after the manner of a ball valve."[10] These two cases, in addition to mine, establish, I think, the auscultatory phenomena of this form of valve lesion. The pulsatile condition of the jugular veins, mentioned in my case as occurring with the auricular systole, and the general

(8) *British Medical Journal*, Jan. 28, 1871.
(9) *Clinical Medicine*, 1862, p. 603.
(10) Walshe. *Diseases of the Heart*, 4th edition, p. 375.

systemic venous congestion unaccompanied by physical signs of pulmonary stasis, form special features of this form of valvular defect. The condition of the liver, which was found on post-mortem examination, shows that the systemic venous stasis had existed for some time in my patient. In future, the præsystolic jugular pulsation and the general venous congestion will be found useful confirmatory evidence of the existence of tricuspid stenosis.

X.

ON A CASE OF PLEURITIC EFFUSION, IN WHICH EMBOLISM FOLLOWED THORACENTESIS BY ASPIRATION.

Gentlemen,

A few days ago we stood at the bedside of a patient in Ward 7, from whom we had removed a large quantity of pleuritic fluid by the aspirator. I then called your attention to a remarkable condition which had followed the operation—viz., gangrene of both lower extremities from embolism of the common iliac arteries. I also gave you what I considered to be the explanation of this rare occurrence. Since then the patient has died; and in this lecture I propose to enter more fully into the case, and to show you how thoroughly the post-mortem examination confirmed my theory of the rare phenomena which the case presented.

The patient, a young man aged twenty-six, a draper's assistant, came to Birmingham from South Wales to seek relief for great difficulty of breathing, from which he had suffered for

more than nine months. He stated that he had been strong and healthy up to the beginning of his present illness, which began as an attack of pleurisy of the right side. When I first saw him he was well-nourished, but of a pale and sallow complexion; his breath was short, especially when he attempted to move about. On examination, the right side of his chest was found to be dull all over from copious effusion, which displaced the heart towards the left, and pushed the liver downwards. The temperature of the body was normal, the pulse 84, and the respirations 24 a minute. As he had already been treated actively by diuretics and blisters, I advised him to enter the hospital for more direct treatment. He had some objection to the evacuation of the fluid, and consequently did not apply for admission till some ten days later. In the interval he had become much worse, the dyspnœa had greatly increased, and he had suffered from sharp pain in the right side.

When admitted (February 10) he was scarcely able to walk across the ward, and in bed he could only breathe when propped up in the sitting posture. In the evening the respirations were 28, the pulse-beats 95 a minute, and the temperature in the axilla 100° F. The right chest was almost motionless; the intercostal spaces were obliterated; the percussion-note was

absolutely dull from base to apex; there was no vocal fremitus; and only distant blowing breath-sounds could be heard. The right chest measured seventeen inches and three-quarters, the left sixteen inches and a half. The left lung was healthy. The heart was displaced towards the left, the apex beating an inch outside the nipple-line. The heart-sounds were perfectly normal, and the impulse was natural. The liver was depressed, and its edge could be felt fully one inch below the costal arch. The tongue was but slightly furred, the appetite good, the bowels constipated. The urine was clear and contained a slight trace of albumen, which had not existed when the patient was first seen.

At the time of his admission he had been taking for three days some pills of digitalis, blue pill, and squills, which had failed to produce diuresis. As the bowels were confined, he was ordered a purgative, and the pills were discontinued. When I saw him the next day he had passed a restless night, and was not any better. The temperature had fallen to 98·8°, and the pulse to 88, but the respirations were 28 a minute. His face and lips, however, looked a little dusky, and his expression was more anxious. As the bowels were being freely acted on by the purgative, I deferred the thoracentesis, but left discretionary power with the house-physician to use the aspirator if the

symptoms became worse towards night. On making his evening visit, my colleague Dr. Rickards (then the house-physician) found the man decidedly worse: the dyspnœa had increased, the lips and face were more dusky, the respirations had gone up to 36 a minute, and the pulse to 126. Dr. Rickards considered the patient's state critical, and very properly determined to evacuate the fluid by the aspirator. By means of this instrument, using a No. 2 needle, Mr. B. May, the house-surgeon, drew off nearly 130 oz. of almost clear and very albuminous fluid of specific gravity 1022. As the fluid flowed from the chest into the aspirator, the breathing improved, and the relief felt by the patient increased with every ounce evacuated. During the operation the man had no cough, and experienced no faintness nor any discomfort whatever. When the operation was completed (the whole or nearly the whole of the fluid having been drawn off) the right chest had become fairly resonant except at the base, the respiratory sounds could be distinctly heard, and vocal fremitus felt. The patient expressed his sense of relief, and assumed with ease an almost recumbent posture; he slept quietly through the night.

On my visit the next morning he was able to lie down in bed. His face and lips were no longer dusky, the breathing was quieter, being only 20

a minute; the pulse was 92 and the temperature was 99°. There had been neither cough nor expectoration. The front part of the right chest was resonant, the respiratory sounds were distinct, and the movements in breathing evident. He complained of feeling low, and also of having had some pain across his back, especially on the left side. He had passed only three ounces of urine since the operation (twelve hours). He was ordered three ounces of brandy, and a mixture containing small doses of iodide of potassium. During the day the pains in his back troubled him a good deal, and he passed very little water—only six ounces, which was high-coloured, with a specific gravity of 1050, and loaded with urea. On the addition of nitric acid to test for albumen, the urea formed a solid mass of crystals above the acid. In the evening his pulse was 100, the respirations 26, and the temperature 99·8°. He passed a comfortable night, but in the morning his pulse had risen to 120, and the respirations to 28 a minute. The temperature was 99·5°. He complained of feeling low and weak, and also of the pain across the back. There was some dulness at the base of the right lung, but above the angle of the scapula the chest was fairly resonant, and the respiratory murmur could be distinctly heard, though not so clearly as on the left side; occasional friction sounds were also heard, mixed

with crackling sounds. The left lung was natural. The heart-sounds were clear; the impulse was normal both in position and force. There was no cough. The urine was still scanty, amounting during the twenty-four hours to only eleven ounces, loaded with urea, and containing a small quantity of albumen, but no sugar. A hot linseed-meal poultice was applied over the kidneys, and gr. v. of pil. scillæ co. ordered twice a day. Early in the evening the patient shivered slightly, his temperature rose to 100·6°, and the pulse to 126, while the respirations remained at 28. During the night he was seized with a violent pain in his right calf, which gradually spread all over the limb. The pain became less towards morning, and then he felt that he had no power in the limb, and that it gradually lost sensation.

In the morning (February 14) I found the limb of a wax-like pallor, cold, almost motionless, and at spots insensible to the touch, while at others it was exquisitely painful on pressure. There was no pulsation in the dorsal artery of the foot, in the posterior tibial, popliteal, or femoral arteries. The limb had evidently lost its blood-supply from obstruction of the main artery above Poupart's ligament. The left leg was warm and natural, and its arteries were easily felt. The respirations were 32, the pulse 120, but the temperature had fallen to 98·8°. The man's expression was singu-

larly calm and free from anxiety. The tongue was clean, the appetite fairly good. He was ordered some brandy-and-egg mixture, instead of his brandy, and some carbonate of ammonia and nitrate of potash were added to his iodide of potassium mixture. The limb was enveloped in cotton-wool, and artificial warmth was applied. During the day he shivered slightly twice, and in the evening his temperature had risen to 100·2°, the pulse to 126; the respirations were 32. He passed a restless night from pain in the left leg similar to that previously felt in the right. In the morning we found the left limb pale, cold, and pulseless. The temperature in the axilla was 100·6°, the pulse 120, and the respirations 28. The urine had increased to 19 oz., and the specific gravity had fallen to 1032; it still contained albumen. In other respects there was little change in the general state of the patient. The right leg was much discoloured, and on the morning of the 16th, when I specially called your attention to the case, the right leg was a typical example of dry gangrene, and the left was following the same course. The patient was worse, too, in other respects—his temperature was 101·2°, the breathing was shallow and quick, and he complained of a pain under his left shoulder-blade. On auscultation, a friction sound was heard in that situation, and dulness at the left base was discovered. There

was evidently pleurisy of the left side, in addition to his other troubles. During the day he became worse, and in the evening he was breathing forty times a minute, his pulse was 168, and the temperature 100·4°. The urine, however, had increased in quantity to 32 oz., specific gravity 1025; still containing albumen, but free from sugar.

After this date the daily reports were all unfavourable. The pulse kept very high, the respirations were very frequent, and the temperature, after falling on one morning to 99·4°, rose, and on two evenings reached 105°. During the last four days of his life the pulse kept at 126, and the respirations varied from 32 to 36. The dulness in the left chest increased, and the gangrene of the limbs advanced. The belly became tumid and also slightly tender on pressure; the skin assumed an icteric tinge. The tongue, however, kept moist and fairly clean, and he took his food (chicken, milk, and rice) and the brandy-and-egg mixture up to the day before his death, when he was sick for the first time. After the 22nd his stomach refused all food, and he died quietly on February 23. He was conscious to the last; he suffered from great dyspnœa during the last few days of his life, and was delirious each night. The secretion of urine was restored on the 18th, and kept up from then till his death, varying from

52 oz. to 59 oz.; the specific gravity fell to 1015, the albumen disappeared, and the urine was in all respects healthy.

Such were the chief clinical details of the case. Before we proceed to read them by the light of the disclosures of the post-mortem examination, we may, I think, profitably recall the views expressed respecting the production of these remarkable phenomena. When the patient was admitted I pointed out to you that the history of the case and the physical signs were those of a long-standing pleuritic effusion, which, judging from the man's temperature and general condition was probably still serous in its character. The active treatment which he had undergone ineffectually, told us that removal of the fluid by thoracentesis promised the best chance of cure. The ordinary treatment had failed to remove the fluid and restore the lung to its functions. The mechanical means remained, and the circumstances of the case indicated their use. With this view the patient was admitted, but the urgency of his symptoms necessitated the operation sooner than I had anticipated. It was performed, indeed, not so much to cure his pleurisy as to save his life from the dyspnœa which threatened it. So far it was successful; but when we stood at his bedside on the fifth day after the operation, it was not to consider the value of aspiration, but rather to study

the embolism which had produced the gangrene of the limbs. The day after the operation, the patient, who had been saved from imminent death by the aspirator, was comparatively comfortable; but two points in his case troubled me—the scanty secretion of urine and the character of his respirations. The breathing did not seem so much relieved as it should have been after the evacuation of so much fluid. The following day found both these causes of anxiety still present, and also a quickened pulse-rate. The scantiness of the urinary secretion and the character of the urine were very puzzling, and it was only on the third morning after the operation, when the embolism of the main trunk of the right lower extremity was diagnosed, that the clue to the renal troubles was found. Two days later, when we stood together round the man's bed, and many of you examined the cold, pulseless, discoloured limbs, I told you that you were observing phenomena altogether new. We were confronted by the unforeseen. In the patient before us, after the evacuation of pleuritic fluid by a most perfect and scientific procedure, embolism of both common iliac arteries, and of the renal arteries, had occurred. On this supposition we could explain the scanty urine and the pain in the back, and on this supposition alone could we explain the characteristic condition of the lower limbs. The

embolus had stopped high up above Poupart's ligament on the right side. This we inferred from the non-pulsation in the external iliac artery and the absence of any attempt to restore the nutrition of the upper part of the limb by collateral circulation; and, if high up, there was no spot more likely than the bifurcation of the common iliac artery.

But the questions naturally occurred to you, as they had to me—Whence these emboli, and what connexion had the operation of thoracentesis and the consequent expansion of the compressed lung with their formation or dislodgment? The possible answers to these questions we discussed together. The emboli must have been derived from some spot between their sites of impaction and the radicles of the pulmonary veins. No clot formed in one of the systemic veins could have run the gauntlet of the pulmonary capillaries and produced all these phenomena. The great vessels coming from the heart presented no condition to account for the formation of a thrombus. The heart-sounds were from the first perfectly healthy, and therefore we could hardly assume that any fibrinous vegetations had been detached from the aortic or mitral valves. Failing these, I next thought of the left auricle as the possible manufactory of the original thrombus. It occurred to me that the heart, displaced by the effusion, might

have been so pressed upon that in some corner of the left auricle near the entrance of the pulmonary veins of the compressed lung, coagulation might have taken place. The clot so formed would have been detached when the heart resumed its natural position and the circulation through the right lung was restored, and once detached would have provided emboli to account for the phenomena of our case. This notion I mentioned to you, but I dismissed it as unsatisfactory on account of the slight amount of cardiac displacement, the absence of any cardiac distress after the operation such as a large auricular thrombus becoming loose would have occasioned, and lastly on account of the intervals which separated the appearance of the embolic phenomena.

The heart and its valves and the great arteries offering us no source for these clots, whence then did they come? One possible origin yet remained —the pulmonary veins. Coagulation in the veins of the affected lung seemed to be the most likely source of the emboli.

The compression of a lung by pleuritic effusion offers conditions favourable to the coagulation of blood in the branches of the pulmonary veins. It is quite conceivable that a lung not completely compressed might have some of its veins full of stagnant blood—stagnant because the pressure from the pulmonary arteries no longer aided its

propulsion into the left auricle. In the case before us the partial and long-continued compression of the lung, before the supervention of the urgent symptoms which forced the man into the hospital, offered conditions most favourable to such thrombosis. At least, so it appeared to me, and therefore concluded that the emboli were most probably formed in the pulmonary veins of the right lung.

When the pleuritic fluid was drawn off and the lung expanded, the blood once again found its way from the right heart along the pulmonary arteries; under this blood-pressure, the coagula in the corresponding veins were gradually detached and swept into the left auricle, whence the blood-current carried them on their fatal course to the renal and iliac arteries. The pulmonary veins in which the smallest clots existed no doubt first became pervious, while the larger and more extensive clots became detached later; hence the intervals which separated the impaction of the emboli. Such was the explanation, founded on the clinical features of the case. This theory seemed the only one which adequately answered the questions as to the source of the clots and the relation of their impaction to the removal of the fluid from the pleura. Let us now turn to the post-mortem facts, and see how they supported the bedside reasoning.

The autopsy was made seventeen hours after death by Dr. Carter, the resident Pathologist. The rigor mortis was well marked; the lower extremities were black, dry, and gangrenous as far as the knees, but less discoloured above. There were traces of blebs having formed on the left thigh. The brain and its membranes were normal in every respect; there was no atheroma nor plugging of the arteries at the base of the brain. The right pleura contained some five or six ounces of fluid, turbid, but not purulent. Both the parietal and visceral surfaces of the pleura were covered with a thick irregular layer of yellowish lymph. This layer was thickest at the anterior borders of the lung, and was cheesy in spots. The right lung was covered by this thickened pleura, and appeared not to perfectly fill the chest at the lower part: it weighed eighteen ounces, was tough, congested, but crepitant, and no part sank in water. The left pleura was firmly adherent at the apex. There were some more recent adhesions at the base, and in the cavity there was about a pint of turbid serous fluid. The pleura was covered with a uniform layer of recent lymph, which was easily stripped off, disclosing the surface studded with miliary tubercles, especially numerous on the opposed aspects of the upper and lower lobes. The left lung weighed also eighteen

ounces, was less tough than the right, of a reddish colour, and congested throughout. The bronchial mucous membrane was congested and covered with a thick layer of mucus. There were no miliary tubercles in either lung.

There was a slight excess of fluid in the pericardium. The heart weighed eleven ounces, was normal in shape and size, and its valves, orifices, and substance were perfectly healthy. Both sides were nearly empty. The right ventricle contained a small recent clot. The left ventricle contained three branched clots, which divided dichotomously, and were apparently casts of some bloodvessels. These clots were loose and unattached in the ventricle; and on opening the auricle a similar firm, branched, pale-coloured clot was seen protruding from one of the pulmonary veins of the right lung. The inner coat of the aorta showed spots of slight fatty change. The descending thoracic and abdominal portions of the aorta were healthy. The common iliac artery on each side was filled by a firm clot; the corresponding veins were quite patent. The embolus on the right side was composed of two parts—the one softer and redder, the other paler and more dense. The pale, tough portion had formed the original embolus, and was caught on the spur at the angle of the division of the common iliac artery, and projected into both

branches, but more especially into the external iliac. On this original embolus the remainder of the clot had been formed by coagulation. In the left iliac artery the whole of the clots were softer and redder than on the right side. It was, however, composed of two portions as on the right side: the original embolus, though smaller, having the same characters as that in the opposite artery. The clots extended upwards on both sides to within about a quarter of an inch of the bifurcation of the aorta, and downwards into both internal and external iliac arteries.

The peritoneum was covered with scattered miliary tubercles, similar to those on the pleura. Some in the mesentery were as large as peppercorns, each surrounded by an areola of congestion, and themselves blackish and pigmented. The smaller tubercles were almost invariably connected with the peritoneal bloodvessels. The intestines on their outer surface were blackish in colour, had lost the healthy peritoneal smoothness, and were congested. There was no excess of fluid in the peritoneum; and except the generally increased vascularity, and the miliary tubercles, no sign of inflammatory change. The stomach and intestines were healthy. The liver weighed sixty-one ounces, was unaltered in size and shape, but presented on section a greenish hue. The gall-bladder was full. The spleen contained a

hæmorrhagic infarction as large as a small pear, wedge-shaped, with the base directed outwards, and with a hard-defined margin. The infarction was in part yellowish in colour, and generally of a lighter shade than the surrounding parenchyma. The kidneys were both enlarged; the left weighed nine ounces and the right seven and a half. On the left were seen several yellowish-red roundish elevations, ranging in size from a pin's head to a small nut. On section, these were found to be the bases of conical hæmorrhagic infarctions from embolism of the branches of the renal artery, and were surrounded by a dark areola of congestion. The right kidney presented only two similar hæmorrhagic spots, but was congested. In other respects, the structure was healthy in both kidneys. The retro-peritoneal glands were unusually large, red, and vascular.

Such is a condensed account of the rich pathological study which the post-mortem examination in this case afforded. You have already seen all the morbid parts, and had a full description of them from the Pathologist. I need not speak of them further; but would call your attention to some few points of clinical interest, such as the seats of the several emboli, the occurrence of the miliary tuberculosis, the discoloration of the skin observed during life, and the pigmentation of several parts found after death.

The theory of embolism by which the chief phenomena of the case were explained found a most complete verification in the post-mortem facts. Not only were the emboli found as was anticipated, but their origin in thrombosis of the pulmonary veins of the compressed lung was most completely established. The presence of the branched clots in the left ventricle, and the lucky discovery of an old tough clot similar to the obstructing emboli in the iliac arteries, protruding from the pulmonary vein, conclusively demonstrated the truth of the theory. The embolic process was beautifully illustrated by the several specimens which you have examined from this case. The hæmorrhagic infarctions of the spleen and kidneys, and the necrosis of the tissues of the lower limbs, form together an unusually complete picture of the results of this process. When emboli enter the circulation from the left heart, they have certain preferences in the course they follow. Whirled along by the blood-current, the little clots commonly pass by both the innominate and other branches of the arch of the aorta, to seek their resting-place in the splenic artery, which is the most frequent seat of impaction. Next, the renal arteries are most frequently obstructed, and then come the iliacs, especially the left. This order of preference was followed in the case we are considering, as far as

the splenic and renal arteries were concerned. The hæmorrhagic infarction in the spleen presented, in its alterations of colour and consistence, evidence of a somewhat longer existence than those in the kidneys; and we are justified in saying that the splenic artery received the first embolic plug from the pulmonary veins of the right side. The renal arteries had not long to wait, and, as we know from the symptoms connected with the kidneys, the blocking of the renal arteries took place—partly at least—in the twelve hours immediately following the thoracentesis. Some forty-eight hours later the right common iliac received its obstructing clot, and the left iliac, which is more usually obstructed than the right, had twenty-four hours longer respite. Some of the infarctions in the kidney occurred no doubt later still, although I think, from their appearance and from the restoration of the urinary secretion, that none were formed for six days before death.

The occurrence of embolism of the renal artery was once diagnosed by the clinical sagacity of Traube, in a case of ulcerative endocarditis, in which albuminuria and hæmaturia supervened. In the present case, the renal pain and the excessively small urinary secretion first attracted our attention; and when the obstruction of the right iliac occurred, the key to the explanation

of these phenomena was given. The existence of a trace of albumen in the urine of our patient before the thoracentesis, and the absence of hæmaturia throughout, made the diagnosis less clear. In this case the renal embolism was associated with pain in the back, and a very scanty secretion of high-coloured, dense urine, which owed its density to the large quantity of urea it contained. There was also an increase in the quantity of albumen, but there was no hæmaturia perceptible to the naked eye, and no record was made by the House-physician of the presence of any blood-cells when he examined the urine microscopically.

Before passing away from this part of the case, I must refer to a question that naturally suggests itself as to the causes which produced the thrombosis in the right pulmonary veins. It is certain that this coagulation does not happen frequently, and I can offer no explanation of its occurrence in this case beyond the condition of the lung. From the history of the patient, it seems to me that the lung had partly expanded after its first compression, and had been compressed again by an increase in the amount of effusion prior to the patient's admission to the hospital. In these conditions of long-continued partial compression, it is possible, I think, that coagulation may have been favoured in some branches of the pulmonary

veins, which, receiving no blood from the corresponding pulmonary arteries, may have yet been filled by a reflux from the larger pulmonary veins. That such thrombosis, however it was produced, is very rare, is proved by the infrequency of such accidents after thoracentesis as those described in this case. Rare as it undoubtedly is, there are conditions yet to be discovered which account for its occurrence. This case has taught us the fact; others must demonstrate the conditions of its production.

Let us now pass on to consider the miliary tubercle. "Pleurisy is a disease full of surprises," and there was yet another surprise for us in the discovery of the tuberculosis after death. The man's temperature when he was admitted, and for some days after the use of the aspirator, indicated no mischief of this kind. The tubercle could hardly have been latent, but was most probably developed after the operation by the absorption of the cheesy matter from the right pleura, or possibly by the absorption of septic matter from the lower limbs. The condition of the patient when the embolism of his common iliacs occurred was so hopeless, and the production of blood-poisoning by absorption of septic matter from the lower limbs so possible, that the pleurisy was referred to this source. It proved, however, to be tubercular, and the cheesy degeneration of the

old false membrane on the right pleura, which was not itself tubercular, was no doubt the source of the infecting matter. The absorption was favoured by the expansion of the lung and the restoration of the circulation through it.

The eruption of tubercles on the peritoneum was a still later event. The tubercles bore unmistakable evidence in themselves of the date of their development. Wherever we examined these little bodies on the omentum, on the intestines, or on the mesentery, they were abundantly pigmented. This pigment, which was so freely deposited in parts of the peritoneum as to give it a dusky hue, was no doubt derived from the destruction of the large mass of blood in the lower limbs, and the consequent setting free of the colouring matter of the red corpuscles. In several specimens of blood-clot taken from the heart and great vessels the microscope detected numerous irregularly-shaped granules of pigment. To this loading of the blood with pigment we may refer the discolouration of the skin, which was similar to the icteric tinge observed in pyæmia and other analogous conditions. The liver, too, in its darkish-green colour and generally pigmented condition, testified to the amount of blood-pigment which it was called upon to transform. Wherever the red blood-cells have their birth-place, there is no doubt that in the liver

they have a burial-place, and that in this organ their pigment is transformed and applied to other uses. In the spleen the pigmented condition was also very marked.

Lastly, I would say a word or two on the treatment in this man's case. We have one regret —that the evacuation of the fluid was not attempted earlier. Months before he came under our care it might have been performed, most probably with success. When the aspirator was used, it was to avert imminent death. So far the paracentesis was successful. The patient was saved; nay, more, for two days he enjoyed more comfort than he had done for weeks, and he lived many days longer than he could have done without the operation. The morbid phenomena which followed had, as far as we can see, no necessary connexion with the time or mode of the evacuation of the fluid. The clots in the pulmonary veins had certainly existed for some time; and whenever and however the circulation in the lung was restored, these clots would have found their way into the aortic system. The withdrawal of so large a quantity of fluid by the aspirator, at one operation, could have had no influence on the after-progress of this case. The man felt so much relief from every ounce withdrawn, and was so free from all the bad symptoms which are laid down as following the evacuation of too large a

quantity of fluid, that the operation was completed. In another case I should certainly not evacuate the whole contents of the chest; it has, indeed, never been my practice to do so. Still, I cannot refer the morbid sequence in this case to the mode of operating: the evacuation of a smaller quantity would, I believe, have been followed by the same occurrences. It is these occurrences which I wish to impress on you. It is always an unpleasant task to point out a new source of danger following a common and important operation, but it is nevertheless a duty. In calling your attention to this case, I have warned you of the possibility of a like occurrence in the future, and I trust I have prepared you, whenever you meet a similar case, to study it with profit to yourselves and advantage to our art.

APPENDIX.

NOTE ON THE PATHOLOGY OF DUCHENNE'S PARALYSIS.

Some few weeks after the sheets containing the lecture on Duchenne's paralysis had been printed, an important addition to the pathology of the disease was made known in a paper read at the Royal Medico-chirurgical Society (May 26th, 1874), by Drs. Lockhart Clarke and Gowers. This paper contained an account of the post-mortem examination in an advanced case of the disease, and of the microscopical appearances discovered by Dr. Lockhart Clarke in the spinal cord. The latter were described in the published abstract of the paper as follows: "The brain and medulla oblongata and meninges of cord were healthy; the spinal cord itself presented various changes throughout the cervical, dorsal, and lumbar regions. The most important was disintegration of the grey substance of the anterior, lower, and central portions of each lateral half. In some places this had occurred chiefly around the vessels, but in others it involved extensive areas, especially in the cervical enlargement, the upper part of the lumbar enlargement, and the conus medullaris. About the level of the last dorsal nerves it had amounted to almost total destruction of the grey matter on each side between the posterior vesicular columns and the

intermedio-lateral tract. Other changes were disintegration of nerve-roots, commencing sclerosis of the lateral and posterior columns, destruction of the white commissure in various places, dilatation of vessels, and extravasations." This description, taken together with the statement made by Dr. Lockhart Clarke at the meeting, that he had some four years ago satisfied himself of the existence of granular degeneration of the cord in another case, gives a new interest to the observations of Barth and Kesteven, who had previously described less decided changes in the nervous centres. Hitherto the pathology of Duchenne's (pseudo-hypertrophic) paralysis has rested on the negative results of post-mortem examinations: the positive facts now announced on the authority of so eminent an observer as Dr. Clarke, render it highly probable that the origin of the disease must after all be sought in the spinal cord.

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